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TRANSMEATAL TYMPANOTOMY*

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The emergence of transaural tympanotomy as an invaluable aid towards improving certain types of hearing must be attributed to the pioneer work of Julius Lempert[1] on tympanosympathectomy. His method was subsequently used by Rosen[2] for surgery of the stapes. Its value lies in the fact that it has opened up new fields in otology by allowing, simply and easily, the inspection of the contents of the middle ear.

Technique. Rosen's method[2] is carried out under local anaesthesia under magnification supplied by the Zeiss otoscope. The circumferential incision begins posterior to the edge of the drum at 6 o'clock, 6 mm. from the annulus, and ends at 12 o'clock. The fibrous annulus is elevated from the sulcus tympanicus. Care must be taken to prevent perforation of the drum or injury to the chorda tympani. The posterior half of the tympanic membrane is folded forward. The contents of the middle ear are now exposed. Not infrequently the stapes is obscured by the posterosuperior bony rim, which must be removed.

Indications. This technique opened up a field of surgery for the treatment of middle-ear deafness which was new to otologists. The main indication is otosclerosis. It must be clearly understood that stapediolysis has not displaced the fenestration operation. Altman points out: 'The recently revised operation of stapes mobilization is in the midst of rapid changes and development. It is not possible to predict how far it will replace fenestration as the preferred surgical treatment for stapes fixation'.

Stapediolysis obviously has many advantages over fenestration. It is a minor surgical procedure and can be performed in quite elderly people, and in cases of unilateral otosclerosis, where fenestration is rarely advised. It may also eliminate the conductive loss in cases of small air-bone gap. Moreover, the patient is hospitalized for only 24 hours. Nevertheless, it cannot be over-emphasized that mobilization is a preliminary procedure and it is advisable to prepare the patient psychologically for failure.

Stapediolysis has captured the imagination of otologists throughout the world. Various techniques are being evolved. The percentage of cases which have improved is slowly mounting, but no standard method has been devised. Lindsay[3] rightly says, 'The histopathology of otosclerosis is the one factor of stapes-ankylosis which does not change, but as the years go on we are more aware of the many variations which exist'. The tendency is at present to divide the cases as follows, according to the degree of pathology present and the resultant fixation of the stapes. Shea[4] and Belluci[5] classify their cases on almost similar lines:

Class 1. There is no narrowing of the stapediovestibular joint; the footplate is blue; palpation of the anterior end of the stapes reveals some degree of fixation but the posterior end is movable. Here the indirect method of stapediolysis is applicable. Pressure with a needle is applied through the lenticular process of the incus *via* the incudostapedial joint; or, failing this, through the head or neck of the stapes. These methods are usually successful.

Class 2. Where there is narrowing of the stapediovestibular joint, but not involving the anterior or posterior crus, the footplate is blue and, owing to the flexibility of the stapes, slight motion can be seen on palpation at one end of the footplate. At the anterior end there is definite fixation, and sometimes the whole footplate is involved. Here the mobilizing forces have to go through the lesion, causing some degree of trauma. In this category the percentage of refixation is high.

Class 3. In this group, if advanced otosclerosis should occur involving the anterior crus, the footplate and the vestibular wall, then mobilization is performed by means of the anterior crurotomy of Fowler.[6] The anterior crus is divided above the otosclerotic lesion, and the footplate is fractured; sound is now transmitted to the oval window through the posterior part of the footplate and the remaining crus.

Class 4. Where there is a large otosclerotic mass present involving the whole footplate or where both crura are fixed by the otosclerotic lesion, the obvious treatment would be to perform the fenestration operation. But Shea,[4] in the USA, and Portmann and Claverie,[7] in France, have developed a new and bold approach. The footplate is removed, preferably *in toto*. Both crura of the stapes are divided and the stapes is displaced out of the operative field. The incus is left *in situ*. A vein graft is taken from the back of the hand. The periosteum is removed for about 2 mm. on all sides of the oval window. The graft is placed with adventitia towards the fenestra. If the stapes has not been disorganized, the head of the stapes is fitted to the lenticular process of the incus and the posterior crus rests on the vein graft, slightly invaginating it. If the stapes is damaged, then a polythene tube is used as a columella. The original ossicular-chain principle is now re-established and the results claimed are good.

Dangers and Complications in Transmeatal Tympanotomy

The dangers in this operation are obvious and manifold.

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The following are the most probable complications:

1. Tearing of the tympanic membrane, where a large perforation is present. A myringoplasty may be required to restore good hearing.

2. Damage to the chorda tympani. Loss of taste and numbness are felt on that side of the tongue, but these lessen and disappear after 3 - 4 months.

3. Dislocation of the incudostapedial joint. Apparently this may be done with impunity. However, one question arises: Does the blood supply to the lenticular process of the incus go through this joint and will necrosis ultimately occur? This point is referred to below.

4. Fracture of the head or neck of the stapes may occur; also fracture of both crura, or fracture of the footplate with depression of a fragment into the vestibule.

5. Haemorrhage may be severe when the surgeon operates near the footplate.

6. Labyrinthitis sometimes follows when he works on the footplate or chisels it out of an otosclerotic mass.

7. Facial paralysis is possible during local anaesthesia but does not last long. It sometimes occurs if the Fallopian canal is prominent and bulges over the oval window, and is even more likely if there is a dehiscence in the bony Fallopian canal.

Anomalies Encountered in the Operation

Transaural tympanotomy has not only made us more familiar with the anatomy of the middle ear but has demonstrated that anatomical variations and anomalies occur not infrequently and may be responsible for many cases of middle-ear deafness. Apart from defects associated with grosser changes, here are a few of the commoner anomalies, which may be very disconcerting when the contents of the middle ear are exposed and the operator is not aware of these anatomical variations:

1. Fixation of the malleus, incus and stapes or fixation of the incudostapedial joint sometimes occurs.

2. The crura of the stapes may be infantile (i.e. large and bulky) or they may be very delicate and break easily.

3. The stapedial tendon may be absent.

4. Abnormal bars of bone may be found.

5. The oval window may be hidden between the promontory and the Fallopian canal. In that event mobilization of the footplate is impossible.

6. Duplication of the round windows, and variations in size, may occur. The round window may be completely occluded by otosclerotic bone. The loss of hearing is then comparable to that with occlusion of the oval window. Strands of mesenchymal tissue may form bizarre patterns over the round window.

7. Erosion or necrosis of the lenticular process of the incus is not uncommon with a normal tympanic membrane. This may well have a bearing on the question mentioned above concerning the possibility that necrosis might result if the blood supply to the lenticular process were jeopardized.

8. Dehiscence of the middle ear, allowing the jugular bulb to herniate, or a glomus jugulare tumour, may be found.

9. Dislocation of the incus may occur in a fracture of the skull, giving rise to conductive deafness with or without facial palsy. It may also follow operation on the mastoid bone. (Recently I had an interesting case of a patient aged 20 who had undergone a cortical mastoid operation 12 years previously. Bone conduction was good; air conduction showed a loss of 50 decibels. At operation it was found that the incus had been dislocated. This was rectified and hearing was restored.)

Other Indications

Congenital fixation of the stapes footplate is an interesting condition which may be diagnosed by clinical means. Shambaugh[8] has described 5 cases and many more are recorded. The key to diagnosis is absence of progression of the hearing loss. Children with a history of purely conductive non-progressive impairment of hearing, in whom the otological findings are essentially normal, may be diagnosed as cases of ossicular discontinuity or congenital ossicular fixation occurring at the stapes footplate. A flat audiometric curve of 50 decibels of pure conductive loss is very suggestive of congenital fixation of the stapes footplate. The uniform bony fusions may be thick or thin. If thin, the footplate is readily mobilized; if thick, fenestration may be advisable.

The transaural approach may also be used for persistent recurrent serous otitis media. The tympanic membrane is elevated and, if necessary, a small polythene tube can be left *in situ* for a few months. Transaural tympanotomy is also usually performed during examination of the ossicular chain preceding tympanoplasty or myringoplasty, and simplifies cases of facial decompression of the transverse part of the nerve. The transaural approach is ideal for cases of labyrinthotomy for Ménière's syndrome, which nowadays may be performed through the foramen ovale, and not, as previously, by opening the mastoid bone. The stapes is displaced, and replaced after destruction of the labyrinth.

The comparative ease with which direct inspection of the middle ear contents may be performed has given considerable stimulus to the treatment of conductive deafness. It now rests with the otologists to ensure that overenthusiasm does not cause these methods to fall into disrepute. Major transmeatal procedures should be undertaken only by operators experienced in cadaver surgery of the middle ear and used to working through a microscope.

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South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

LIVE ATTENUATED POLIOVACCINE

The mass campaign of vaccination with Sabin Type I poliovaccine is underway in South Africa. Towards the end of October the Department of Health released two million doses of this vaccine for use by local authorities in the larger centres. In April and May 1961 further batches of all three types of vaccine will be issued. More than a million doses of the vaccine produced at the laboratories of the Poliomyelitis Research Foundation have already been used with good results in Kenya and Mauritius.

South Africa has thus joined many other countries in an all-out attempt to banish poliomyelitis completely. This long-desired achievement in the field of public health will be brought about if the Sabin strains of poliovaccine can indeed give life-long immunity, as is expected. In a recent issue of the *Journal* Prof. R. Turner[1] gave a comprehensive review of the research and field trials leading to the adoption by many countries of the Sabin strains of oral poliovaccine. He pointed out that, even before the inactivated vaccine perfected by Salk was developed, many authorities believed, on general principles, that the best hope of a really efficient prophylactic against poliomyelitis lay in the production of a live vaccine.

Even the great success of the Salk inactivated poliovaccine did not deter these research workers, foremost among whom was Koprowski. By 1957 several strains of live poliovaccine had been developed and were ready for large-scale trials. Three different strains were selected, namely, the Koprowski, the Cox, and the Sabin strains.

Turner, in his article, discussed in detail the results of various trials with these strains, and made it quite clear that all the evidence to date showed that Sabin strains were both the safest and the most effective. These are the strains that have been used in about sixty million Russians and in millions of other subjects with no ill-effects whatsoever. Turner does not believe that there is as yet sufficient evidence for a final opinion on the safety and effectiveness of the Koprowski strains (although they have shown themselves so far to be apparently safe and effective), and he feels that no definite decision has been reached regarding the safety of the Cox strains, although they are also apparently effective. All the evidence, therefore, goes to show that, in this country, we are producing the safest and most effective live poliovaccine available.

Turner emphasized an important public-health point in his article. He says: 'Once we have embarked on this course (the administration of live poliovaccine) there can be no turning back. If polioviruses are banished from the environment, no child will be able in future to acquire a natural immunity by normal and accidental infection. Feeding of all newborn children with live vaccine at the earliest age then becomes essential for, should they grow up without an immunity, and the polioviruses return, disaster will inevitably follow'.

This is a facet of the problem that will have to be borne in mind by all public-health authorities. At present, a large proportion of the population develops a natural immunity to poliomyelitis by early adulthood. We may be able to wipe out poliomyelitis completely in South Africa, but it may again be introduced from some other country. Unless all children are immunized in infancy against poliomyelitis, as they are against smallpox, they will be at great risk if wild strains of poliovirus reappear. It may be necessary to institute compulsory vaccination against poliomyelitis in the future to obviate the possibility of a catastrophe.

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VOORSKRIFTE VIR MIDDELS WAT POTENSIEEL SKADELIK IS

In die uitgawe van die *Tydskrif* van 26 Maart 1960 was 'n byvoegsel ingevoeg waarin die reëls wat handel oor die opstel van voorskrifte vir middels wat potensieel skadelik is, uiteengesit word. Hierdie dokument is gesamentlik deur die Mediese Vereniging van Suid-Afrika en die Farmaseutiese Vereniging van Suid-Afrika opgestel vir die leiding van dokters.

By die laaste kongres van die Farmaseutiese Vereniging van Suid-Afrika is melding gemaak van die feit dat baie dokters nog steeds die voorskrifte van die Geneeskundige-, Tandheelkundige- en Aptekerswet oortree deur die manier waarop hulle hul voorskrifte uitskryf. Ons kan dit nie sterk genoeg onder die aandag van dokters bring nie om, wat hierdie saak betref, reg op te tree nie.

Afdeling 61*bis* van die wet lê die vereistes vir hierdie voorskrifte baie duidelik neer, naamlik dat die voorskrif die volgende informasie moet bevat: die datum waarop die voorskrif gegee word; die naam en adres van die pasiënt; die naam en hoeveelheid van die middel wat verskaf moet word; die aantal kere en die periodes waarop die voorskrif uitgevoer moet word en, behalwe in die geval van preparate vir eksterne gebruik, die hoeveelheid en veelvuldigheid van elke dosis wat geneem moet word; en die gewone handtekening, adres, en professionele kwalifikasies van die dokter wat die voorskrif uitgeskryf het. Die voorskrif moet in die dokter se eie handskrif wees.

Hierdie vereistes word baie dikwels nie nagekom nie deurdat die een of ander puntjie van die vereiste informasie weggelaat word. Een geval van so 'n weglating, byvoorbeeld, wat baie moeite vir die apteker veroorsaak en gevolglik ook vir die dokter, is die sterkte van die dosis van etiese preparate. 'n Middel is byvoorbeeld soms verkrygbaar in sterktes van 25, 50, of 100 mg., maar die dokter skryf dikwels net ‚20 tablette' voor. Dit is dan vir die apteker nodig om die dokter op te lui om hierdie fout te herstel, en dit is dikwels nie maklik om die dokter in die hande te kry nie. Baie tyd en moeite sou dus vir die apteker sowel as vir die dokter bespaar kan word as hierdie punt in gedagte gehou word by die skryf van voorskrifte.

'n Pligsgetroue apteker, wie se plig dit is om nie middels te verskaf op grond van 'n onvolledige of verkeerde voor-

skrif nie, sal die dokter versoek om die voorskrif oor te skryf in terme van die wet. Sommige dokters word prikkelbaar oor sodanige versoeke en weier om hulle voorskrifte te verander; andere weer is te besig om dit dadelik te doen en die saak word dan daar gelaat om maar net vergeet te word in die loop van die drukte van 'n besige praktyk. Al hierdie dinge dra nie daartoe by om 'n gelukkige dokter-apteker verhouding te bewerkstellig nie, heeltemal afgesien van die straf ('n boete van nie meer as £100 nie of gevangenisstraf vir 'n periode van nie langer as ses maande nie sonder die keuse van 'n boete, of sowel boete en gevangenisstraf) waaraan die dokter en die apteker altwee blootgestel is.

'n Ander aspek van die probleem van die uitreiking van voorskrifte vir middels wat potensieel skadelik is, spruit voort uit telefoniese instruksies. Die wet lê neer dat 'n apteker hierdie middels mag verskaf in gevalle van nood op telefoniese instruksie deur 'n dokter wat die apteker persoonlik ken. 'n Voorskrif in die voorgeskrewe vorm wat hierdie instruksie dek, moet egter nog altyd binne vier-en-twintig uur deur die dokter aan die apteker gestuur word.

Ongelukkig is dit die geval dat dokters dikwels gebruik maak van die vergunning om instruksies van hierdie aard per telefoon te gee, maar hulle versuim net so dikwels om die geskrewe voorskrif aan te stuur. Die Suid-Afrikaanse Aptekersraad, wat dieselfde statutêre magte oor aptekers het as wat die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad oor dokters het, is van mening dat dit nie die verantwoordelikheid van die apteker is om die dokter te verplig om 'n voorskrif te stuur wat die telefoniese opdrag dek nie. *Die dokter* is verantwoordelik om toe te sien dat dit gebeur.

Die sesde skedule van die Geneeskundige-, Tandheelkundige- en Aptekerswet bevat 'n lys van middels wat potensieel skadelik is. Die lys word van tyd tot tyd gewysig deur byvoegings en weglatings, byvoorbeeld antihistamiemiddels wat eertyds op die lys was, is nou nie meer daarop nie. Die lys bevat egter nog sulke middels soos sulfonamiedes en antibiotiese middels, barbiturate (behalwe wanneer hulle in baie klein hoeveelhede voorgeskryf word), hormone, rauwolfia-preparate en antistollingsmiddels. Soos dus gesien kan word, bevat 'n groot aantal moderne voorskrifte die een of ander middel wat potensieel skadelik is.

Die gemiddelde dokter kan nie verwag word om op die hoogte te bly van al die wysigings wat dikwels ten opsigte van hierdie lys gemaak word nie, afgesien nog van die feit dat die name wat in die lys voorkom die farmaseutiese name van die middels is waarmee die meeste dokters nie bekend is nie. Ons wil dus aan die hand gee dat dokters 'n gebruik daarvan moet maak om alle voorskrifte op te stel soos wat aangedui is, met ander woorde, *asof alle middels potensieel skadelik is*. (Voorskrifte vir gewoontevormende middels val natuurlik in 'n spesiale klas — in hierdie geval moet al die genoemde informasie op die voorskrif verskyn en daarby is daar ook nog die voorwaarde dat die voorskrif net een maal uitgevoer kan word).

As dokters alle voorskrifte uitskryf soos vereis deur afdeling 61*bis* van die wet sal aan alle wetlike vereistes voldoen word en baie nietige prikkelings sal voorkom word. Ook sal dit verhoed dat onaangenaamheid tussen dokters en aptekers ontstaan. Dokters sal minder deur aptekers lastig geval word in 'n poging om hulle sover te kry om die voorskrifte te verander, en hulle sal dan ook seker kan wees dat hulle geen wet oortree nie.

PURULENT MENINGITIS IN 204 BANTU CHILDREN

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With the advent of penicillin and the broad-spectrum antibiotics it was hoped that the problem of therapy for acute purulent meningitis was about to be solved. However, the diversity of present therapeutic regimens indicates that there is still no entirely satisfactory approach. Furthermore, with similar treatment, the cure rate varies in different reports. In the following analysis we have attempted to show that the prognosis of acute purulent meningitis is affected not only by the dosage and mode of administration of antibiotics, but also by certain clinical features which develop during the course of the illness. Against this background, we have tried to evaluate a number of therapeutic regimens used at this and other hospitals.

Material and Methods

The series consists of 204 consecutive cases of purulent meningitis in Bantu children under 10 years of age admitted to this hospital from a poor socio-economic environment during the period 1 January 1954 - 31 July 1958. The cases are classified as follows according to the bacteria that were found: Pneumococci 54, *Haemophilus influenzae* 32, meningococci 49, other organisms 6, no organisms found 63.

In 141 cases the diagnosis was made by demonstrating the causative organism in the cerebrospinal fluid (CSF) either microscopically or on culture. In the remaining 63 cases no organism was either seen in the CSF or cultured, and the diagnosis was made on the clinical condition and the cytology and biochemistry of the CSF, which suggested a bacterial rather than a viral aetiology.[50,51] Tuberculous meningitis was excluded, and so were relapses after an apparently satisfactory remission.

In all but 2 cases the diagnosis of meningitis was made on admission after a lumbar puncture. The exceptions were: (1) a 2-weeks-old infant with severe bronchopneumonia and suspected congenital heart disease, in whom pneumococcal meningitis was found at autopsy; and (2) a 7-months-old girl admitted in status epilepticus, in whom the CSF was found to be normal on 2 separate occasions (lumbar puncture and cisternal puncture), yet autopsy revealed an acute purulent meningitis of unknown aetiology.

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The organisms isolated from the CSF were tested by the disc method (Baragwanath modification[1]) for sensitivity to penicillin, streptomycin, chloramphenicol, tetracycline, oxy- and chlortetracycline, and sulphonamides.

Each treatment group contained cases of all degrees of severity and there was no selection of cases for different therapeutic regimens. Patients who died within 6 hours of admission to hospital were excluded from the evaluation of treatment.

The nutritional state of each child was assessed. In this we were guided by the body weight and the stigmata of protein malnutrition.[2]

In this paper the term 'focal neurological signs' signifies spasticity, cranial nerve lesions and Jacksonian fits, whereas the term 'convulsions' is reserved for generalized seizures with loss of consciousness. Subdural effusions were looked for in all cases where the clinical progress was inexplicably unsatisfactory. Where found, these effusions were treated by repeated aspiration through the anterior fontanelle. Surgical exploration was not required in this series.

PNEUMOCOCCAL MENINGITIS

The age of the 54 patients with pneumococcal meningitis ranged from 2 weeks to 7 years; 42 (78%) were under 1 year of age. There were almost as many children in the 6-12 months group as in the group under 6 months. There were 29 girls and 25 boys.

All patients received intramuscular (IM) penicillin as follows: 20 cases (group I) received 1 mega-unit hourly, 23 cases (group II) received 1 mega-unit 2-hourly, and 1 patient received 1 mega-unit 3-hourly. This therapy was continued for at least 24 hours and not more than 72 hours, after which the interval was gradually increased, the dose remaining constant. Seven patients (group III), who were all admitted before 1955, received less than 0·5 mega-unit of penicillin per dose, usually every 4 or 6 hours. Three patients died before treatment could be commenced.

Intrathecal (IT) penicillin was given to 25 patients in doses of 10,000 - 25,000 units and 1 patient received 50,000 units on 2 occasions. IT penicillin was instilled from 1 to 10 times, half the patients receiving not more than 2 injections.

Sulphonamides were given to 47 children, in amounts of 2 grains per pound body-weight per day (per lb. bw per day).

Results

Of the 54 patients 33 (61%) survived but 8 (15%) of these remained with neurological sequelae (Table II) (4 with decerebrate rigidity, 2 with hemipareisis, 1 with lower motor neurone palsy of the VIIth nerve and 1 with hydrocephalus).

All 21 deaths in the series ocurred within 72 hours of admission, whatever the mode of treatment; 15 patients died within 24 hours of admission, including 11 cases admitted in coma (Table I); 3 of the deaths occurred within 6 hours of admission. Of the 22 fatalities 17 occurred among the girls and 5 among the boys.

Subdural effusions were found in 8 patients (15%), and hospitalization was prolonged for more than 28 days in 4 of these. All survived and 6 recovered completely. All except 1 were diagnosed within 7 days after admission.

TABLE I. STATE OF CONSCIOUSNESS ON ADMISSION TO HOSPITAL IN 21 FATAL CASES OF PNEUMOCOCCAL MENINGITIS

Survival in hospital	*Fully conscious*	*Comatose or stuporose*	*Total*
Less than 24 hours	4	11	15
24 - 48 hours	1	3	4
49 - 72 hours	1	1	2

Factors of Prognostic Significance

Age. Among the children under 6 months of age 11 (50%) died and 11 recovered, 4 of them only partially.

TABLE II. INFLUENCE OF AGE OF PATIENT ON OUTCOME OF PNEUMOCOCCAL MENINGITIS

Age of patient	*Full recovery*	*Recovery with sequelae*	*Deaths*	*Total*
Under 6 months	7 (32%)	4 (18%)	11 (50%)	22
6 months - 1 year ..	13 (65%)	2 (10%)	5 (25%)	20
Over 1 year	5 (42%)	2 (16%)	5 (42%)	12
	25 (46%)	8 (15%)	21 (39%)	54 (100%)

Of the 20 children aged 6 months - 1 year, 5 died and 15 recovered, 2 with sequelae. Of the 12 patients over 1 year, 5 died and 7 recovered, 2 with sequelae (Table II).

Duration of illness before admission. Table III shows that of the 25 children who had been ill for 2 days or less before admission 15 recovered, 4 with neurological residua; of the 19 children who had been ill 3 - 7 days before admission 15 recovered, 2 with sequelae; and of the 9 patients who had been ill for more than 7 days 6 died and 1 recovered completely.

TABLE III. INFLUENCE OF DURATION OF ILLNESS BEFORE COMMENCEMENT OF TREATMENT ON OUTCOME OF PNEUMOCOCCAL MENINGITIS

Duration of illness before treatment	*Full recovery*	*Recovery with sequelae*	*Deaths*	*Total*
2 days or less	11 (44%)	4 (16%)	10 (40%)	25 (46%*)
3 - 7 days	13 (68%)	2 (11%)	4 (21%)	19 (35%*)
More than 7 days ..	1 (11%)	2 (22%)	6 (67%)	9 (17%*)
Not recorded	—	—	1 (100%)	1 (2%*)

* Percentage of total cases.

Associated lesions. Pneumonia was present in 6 of the 54 children, pertussis and pulmonary tuberculosis in 1 each, purulent otitis media in 8, and advanced malnutrition in 4, of whom 3 died and 1 was discharged blind, deaf and spastic (Table IV).

TABLE IV. INFLUENCE OF ASSOCIATED LESIONS ON OUTCOME OF PNEUMOCOCCAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Acute pneumonia ..	2	—	4	6
Other pulmonary pathology	2	—	—	2
Purulent otitis media ..	7	—	1	8
Malnutrition	—	1	3	4

Neurological manifestations. Of the 25 patients with clouding of consciousness on arrival in hospital 5 recovered completely, whereas of the 29 who were conscious 20 made a full recovery. Convulsions occurred in 29 patients

TABLE V. INFLUENCE OF NEUROLOGICAL MANIFESTATIONS ON OUTCOME OF PNEUMOCOCCAL MENINGITIS

	Complete recovery	*Recovery with sequelae*	*Deaths*	*Total*
Comatose on admission ..	2	1	8	11
Stuporose on admission ..	3	4	7	14
Conscious on admission ..	20	3	6	29
Convulsions with clouding of consciousness ..	4	4	11	19
Convulsions with full consciousness	6 } 10	1 } 5	3 } 14	10 } 29
No convulsions	15	3	7	25

and were distributed among all age groups. Fourteen of the deaths (66% of total deaths) occurred in the patients who had convulsions. (Table V.)

Fever. All 7 patients whose temperature returned to normal within 4 days or less after admission recovered completely. Of those whose temperature remained elevated for 5 - 10 days 13 (76%) did well; 6 of the children with subdural effusions fell into this group. The incidence of neurological sequelae was high in patients in whom pyrexia persisted for more than 10 days (Table VI). The height of the pyrexia on or after admission was of no prognostic significance.

TABLE VI. PROGNOSTIC SIGNIFICANCE OF DURATION OF PYREXIA AFTER COMMENCEMENT OF TREATMENT IN 33 NON-FATAL CASES OF PNEUMOCOCCAL MENINGITIS

Duration of pyrexia	*Full recovery*	*Permanent neurological damage*	*Transient subdural effusions*	*Total*
4 days or less	7	—	—	7
5 - 10 days	13	4	6	17
More than 10 days ..	5	4	2	9

Period of illness in hospital. From Table VII it can be seen that 7 children were clinically well within 14 days, 14 others had recovered by the 28th day; and 7 remained in hospital for more than 28 days, including 4 with subdural effusions and severe neurological sequelae. These 4 were kept in hospital until there was no further improvement and/or until they could take feeds by mouth. One of these patients remained in hospital for 74 days. Only 3 of all these patients relapsed and required re-admission to hospital.

TABLE VII. DURATION OF HOSPITAL STAY IN RELATION TO COURSE OF PNEUMOCOCCAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Subdural effusion*	*Total*
14 days or less	7	—	—	7
15 - 28 days	14	5	4	19
Over 28 days	3	4	4	7

Bacterial Sensitivity to Antibiotics

Cultures from 22 patients were tested for sensitivity to antibiotics. All were sensitive to penicillin and oxytetracycline, all but 2 were sensitive to chloramphenicol and chlortetracycline, and all but 1 were sensitive to tetracycline; 7 strains were completely insensitive to sulphonamides; and 4 were resistant to streptomycin.

TABLE VIII. MODE OF PENICILLIN ADMINISTRATION AND RECOVERY RATE FROM PNEUMOCOCCAL MENINGITIS

Treatment Group	*Full recovery*	*Recovery with sequelae*	*Deaths*	*Total*
I. (*a*) 1 million units IM penicillin hourly ..	7	–	4	11
(*b*) As above + IT penicillin ..	6 } 13 (65%)	1 } 1 (5%)	2 } 6 (30%)	9 } 20
II. (*a*) 1 million units penicillin IM every 2 hours	4	1	7	12
(*b*) As above + IT penicillin ..	4 } 8 (35%)	6 } 7 (30%)	1 } 8 (35%)	11 } 23
III. 1 million units IM penicillin 3 hourly ..	1	–	–	1
IV. (*a*) Low doses of IM penicillin 4 - 6 hourly	2	—	–	2
(*b*) As above + IT therapy	1 } 3	—	4 } 4	5 } 7
V. Death before commencement of treatment	—	—	—	3

IM = intramuscular. IT = intrathecal

Treatment

The effect of penicillin dosage on recovery. Table VIII shows that there was no significant difference in death rate between group I (hourly injections) and group II (2-hourly injections); but the number of complete recoveries in group I was 13 (65%), whereas it was only 8 (35%) in group II. The results in group I(*a*) (IM penicillin only and I(*b*) (IM penicillin and IT penicillin) were similar; but in group II(*a*) there were 7 deaths and 1 recovery with sequelae, and in II(*b*) 1 death and 6 recoveries with sequelae. The latter group received 2-hourly IM penicillin with IT penicillin.

Discussion

Prognosis

Our results show that there are a number of factors which affect the prognosis of pneumococcal meningitis adversely. The most outstanding of these was a depressed level of consciousness on admission, which was found in 48% of cases.

A bad result was also commoner in children under 1 year of age, who comprised 78% of our cases. Where a poor nutritional state coexisted with meningitis the final result was always bad. Other adverse factors of less significance were the presence of pneumonia and a pyrexia which persisted for more than 10 days after commencement of treatment. Results were also poor in children in whom meningitis remained untreated for 7 days or more.

However, patients with an illness of less than 48 hours before admission still had a significant mortality, but those with an illness for 3 - 7 days had the lowest number of deaths. A possible explanation for this difference is that children with overwhelming infection are brought to hospital early, whereas a delay may occur before the parents seek medical advice for those children in whom the disease is less acute. We are unable to explain why the mortality was significantly higher in girls than in boys. This has not been reported elsewhere.

While most of our findings are in agreement with those of other authors,[2-13] there are some points of variance. The incidence of clouding of consciousness and convulsions was much higher in our series than in those of others,[10,14] and this may help to account for our poor results. A small series reported with outstandingly good results had a very low incidence of coma and stupor.[10]

The deleterious effect of malnutrition on the outcome has not been noted elsewhere, but we cannot overlook it because malnutrition is common in children attending this hospital.[15]

Other associated lesions were an infrequent occurrence in our survey in comparison with other reported series.[4,14]

Focal neurological signs, either with or without clouding of consciousness, had little bearing on the outcome, which is in contrast with conclusions drawn by Zilberg.[13] It has often been stressed that the outcome of meningitis may be improved if subdural effusions are discovered and treated early.[6,10,16] This was borne out in our series, and of the 8 patients in whom effusions occurred, 6 recovered completely.

Treatment

As many of the 15 patients who died within 24 hours of admission received energetic treatment, it seems un-

likely that any form of therapy employed would have been effective in these children. If these are excluded from the series the mortality rate becomes considerably reduced.

Penicillin

In this series the pneumococcus was always highly sensitive to penicillin in *in vitro* testing. Unfortunately penicillin does not cross the blood-brain barrier easily[17,18] even in the presence of inflamed meninges.[19] This barrier is penetrated adequately only when the blood level of penicillin reaches a very high peak such as one obtains with an IM injection of 1 mega-unit.[20] Similar levels are not reached with continuous intravenous therapy.[21] Massive IM doses given every 2 hours usually result in CSF levels ranging from 0·08 to 1·25 u./ml.[22] These levels, however, are reached only about 8 hours after commencement of 2-hourly IM injections and this makes it advisable to give an IT injection of 10,000 - 20,000 u. of penicillin at the beginning of treatment.[22] In our series better results were obtained when this initial IT dose was given in addition to 2-hourly IM penicillin (Table VIII, group II*b*).

After an IM injection of 1 million units of penicillin the peak serum level is usually attained within 20 minutes, and the level then falls rapidly in the first hour to a quarter of the peak concentration, and to an eighth in the second hour.[20] Penetration of the blood-brain barrier, therefore, occurs only during a part of this 2-hour period. Thus it seems reasonable to assume that hourly injections would maintain higher penicillin levels in the CSF than 2-hourly injections. We have used this hourly regimen in 20 patients with a marked reduction in the sequela rate (Table VIII, group I).

Complications from this massive penicillin dosage are rare. In a few patients injection abscesses were noted and occasionally oedema of both lower limbs occurs in very small children; allergic manifestations have not been noted in this series. Repeated injections inflict considerable pain in conscious patients and may cause psychological trauma. Nevertheless, this drastic form of therapy is justified by the improved results we achieved.

It might be thought that the type of therapy outlined above could be matched by widely-spaced IM injections and repeated IT instillations of penicillin for many days. This approach has been tried,[5,10,23] but was finally discarded in favour of 2-hourly IM injections without IT therapy.[14]

The rôle of sulphonamides cannot be evaluated from this series, because we lacked controls, since there were only 7 patients who did not receive them. There was a high incidence of *in vitro* resistance to these drugs, which were used mainly for their supposed synergistic effect with penicillin.[24,25]

Broad-spectrum Antibiotics

Of the broad-spectrum antibiotics in use at present chloramphenicol and tetracycline appear to be the most promising in the treatment of pneumococcal meningitis.

Concentrations of chloramphenicol in the CSF have been consistently high after oral and parenteral administration, and in some cases even higher than the serum level.[26] To our knowledge the use of chloramphenicol in pneumococcal meningitis has been limited to only a few patients.[27,28] As its administration is easy and side-effects few,[29] we consider that this drug should be afforded a more extensive trial notwithstanding that two cultures from our cases were resistant to it.

The oral administration of tetracycline, even in high dosage, results in inadequate levels in the CSF[30] and parenteral use is therefore essential.[30] To achieve adequate CSF levels of 0·75 - 3·0 μg./ml., the dosage recommended is 75 mg. per kg. bw per day (initially IM), but this leads to necrosis and abscess formation at the site of injections.[30]. In the only series reported in which this drug was used alone, 22 mg. was administered per kg. bw per day. There was a recovery rate of 73% with 14% sequelae.[13]

Chlortetracycline and oxytetracycline do not cross the blood-brain barrier as readily as tetracycline[26,31-33] and have similar disadvantages.

INFLUENZAL MENINGITIS

During the period under review there were 32 patients with meningitis caused by *Haemophilus influenzae*. Their ages ranged from 3 months to 2 years; 6 (19%) were under 6 months, 17 (53%) between 6 months and 1 year, and 9 (28%) between 1 and 2 years (Table IX). There were 18 boys and 14 girls.

Thirteen patients received both streptomycin (20 mg. per lb. bw per day) and sulphadiazine (2½ gr. per lb. bw per day); and 9 patients received chloramphenicol palmitate, of which 6 were also given sulphadiazine. A further 9 children were treated with a combination of streptomycin, sulphadiazine and chloramphenicol. Chloramphenicol was given to 7 patients in doses of over 60 mg. per lb. bw per day (Table XIII).

Results

Of the 32 patients 28 (88%) survived, but 8 remained with neurological sequelae. All but one of the 4 deaths occurred within 24 hours of admission; the exception was a malnourished child who had a 3 weeks' history of convulsions, remained stuporose in hospital, and died after 7 days. All the fatal cases were 6 - 12 months old (Table IX).

TABLE IX. INFLUENCE OF AGE OF PATIENT ON OUTCOME OF INFLUENZAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Less than 6 months ..	5	1	–	6 (19%)
6 months - 1 year ..	8	5	4	17 (53%)
Over 1 year	7	2	–	9 (28%)
	20 (63%)	8 (25%)	4 (12%)	32 (100%)

Subdural effusions were discovered in 7 (22%) cases; none of these patients died, but 2 were left with neurological sequelae (Table XIII).

Factors of Prognostic Significance

Duration of illness before admission to hospital. Table X shows that of the 13 children who had been ill for 2 days or less before admission to hospital, 11 recovered, 3 with residua. All 10 children who had been ill for 3 - 7 days before admission survived but 3 remained with

TABLE X. INFLUENCE OF DURATION OF ILLNESS BEFORE COMMENCEMENT OF TREATMENT ON OUTCOME OF INFLUENZAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Less than 2 days	8	3	2	13
3 - 7 days	7	3	–	10
More than 7 days ..	5	2	2	9

neurological sequelae. Of the 9 patients who had been ill for more than 7 days 2 died; of the survivors 2 had neurological residua.

Associated lesions. Five children were malnourished, and of these 2 died and 3 remained with residua. Other associated lesions were found in 11 patients and were limited to pneumonia (6) and purulent otitis media (5).

Neurological manifestations. Of the 13 patients with clouding of consciousness on arrival in hospital 3 recovered completely, whereas of the 19 who were conscious 17 made a full recovery. In 18 (56%) of the patients

TABLE XI. INFLUENCE OF NEUROLOGICAL MANIFESTATIONS ON OUTCOME OF INFLUENZAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Coma on admission ..	2 } 3	2 } 6	1 } 4	5 } 13
Stupor on admission ..	1	4	3	8
Conscious on admission ..	17	2	0	19
Focal neurological signs ..	2	2	0	4
Convulsions + clouding of consciousness ..	1 } 8	5 } 6	4 } 4	10 } 18
Convulsions + full consciousness	7	1	0	8
No convulsions	12	2	0	14

convulsions occurred. All the fatalities took place in this group, while only 8 cases (44%) recovered fully. Of the cases without convulsions 11 (78%) recovered completely. (Table XI.) Of the total of 8 patients with neurological damage 5 had decerebrate rigidity, 2 remained with a spastic hemiparesis, and 1 had a flaccid paralysis of an arm. These 8 patients were evenly distributed among all age-groups (Table IX).

Fever. There were 13 patients who became apyrexial within 10 days of admission and only 1 of these had neurological sequelae. In 15 patients the fever persisted for more than 10 days and 7 of these had neurological damage.

Period of illness in hospital. The duration of the patients' stay in hospital is analysed in Table XII in relation to their degree of recovery. Only 3 cases were clinically well within 14 days after arrival in hospital. A

TABLE XII. DURATION OF HOSPITAL STAY IN RELATION TO COURSE OF INFLUENZAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Subdural effusions*	*Total*
14 days or less	3	—	—	3
15 - 21 days	11	2	2	13
More than 21 days ..	6	6	5	12

further 13 patients recovered by the 21st day, among which were 2 patients with subdural effusions and 2 with neurological sequelae. Twelve children remained in hospital for more than 21 days; these included 6 with neurological residua and 5 with subdural effusions. One patient remained in hospital for 68 days; this child had a brain-stem lesion with cortical atrophy. A relapse occurred in 2 children; both were readmitted to hospital and survived, 1 with sequelae.

Bacterial Sensitivity to Antibiotics

Cultures from 18 patients were tested for sensitivity to antibiotics. All were sensitive to chloramphenicol; 3 were resistant to streptomycin, 1 to tetracycline, 3 to oxytetracycline and 4 to chlortetracycline.

Treatment

Of the 13 patients who received a combination of streptomycin and sulphadiazine without chloramphenicol, 3 died and 10 recovered, 3 with residua. Of the 18 patients

TABLE XIII. EFFECT OF TREATMENT ON OUTCOME OF INFLUENZAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Streptomycin + sulphadiazine	7	3	3	13
Streptomycin + sulphadiazine + chloramphenicol	5	3	1	9
Chloramphenicol	7	2	—	9
Tetracycline	1	—	—	1

to whom chloramphenicol was administered either alone or with streptomycin, 1 died and 17 recovered, 5 manifesting neurological damage. (Table XIII.)

Discussion

The incidence of influenzal meningitis at this hospital is lower than that of pneumococcal meningitis;[12] similar observations have been made elsewhere in South Africa.[9,13] Outside this country this situation is reversed.[6,27,34,35] Although the mortality of influenzal meningitis is relatively low, the high incidence of residua still makes it a dreaded disease in the Bantu.

In spite of the small number of cases, some factors of prognostic significance have emerged from this survey. The most important single adverse factor was clouding of consciousness; others were malnutrition, and a pyrexia which persisted for 10 days or more after commencement of treatment. Where convulsions were not associated with clouding of consciousness their presence did not seem to affect the outcome (Table XI).

As reported by others, the incidence of subdural effusions was slightly higher than in pneumococcal meningitis.[16,36,37] The occurrence of these effusions did not appear to affect the outcome adversely.

It was impossible to draw conclusions from the results obtained with the 3 treatment schemes used in the series, because the individual groups were too small. A study of the literature shows that the question of the antibiotic of choice is still undecided.[13,35,38-42] In recent years chloramphenicol appears to have become the most favoured drug,[35] and it is worthy of note that all the cultures from our patients were sensitive to it. The superiority of crystalline chloramphenicol over the esterized form has been pointed out.[26,37,43] Only the esterized form was used in this series, and our results would probably have been better if the crystalline form had been used.

MENINGOCOCCAL MENINGITIS

There were 49 sporadic cases of meningococcal meningitis in this survey; no epidemic of the disease occurred during the period. The ages of the patients ranged from 12 days to 9 years; 10 (21%) were under 6 months of age, 16 (33%) between 6 months and 2 years, and 22 (46%) over 2 years (Table XIV). There were 30 boys and 19 girls.

All but 1 patient received IM penicillin in doses ranging from 250,000 u. 6-hourly to 1 mega-unit hourly. All patients received sulphonamides, usually sulphadiazine, in amounts of 2 grains per lb. bw per day, initial dose usually being given parenterally, and subsequent doses orally.

Results

Of the 49 patients 42 (85%) survived, 4 of these with neurological sequelae.

All 7 deaths occurred within 48 hours of admission to hospital; 5 patients died within the first 6 hours. One of the 2 fatal cases with purpura came to autopsy and the adrenal glands were found to be normal.

Subdural effusions were encountered in 3 patients, who were all less than 6 months of age. None had been ill for more than 2 days before admission to hospital and all recovered fully.

Factors influencing the Prognosis

Age. Table XIV shows that 1 death occurred among the 10 children under 6 months of age, while 9 recovered, 2 with neurological damage. Of the 16 children between

TABLE XIV. INFLUENCE OF AGE OF PATIENT ON OUTCOME OF MENINGOCOCCAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Less than 6 months ..	7	2	1	10 (21%)
6 months - 2 years ..	13	—	3	16 (33%)
Over 2 years	18	2	3	23 (46%)
	38 (77%)	4 (8%)	7 (15%)	49 (100)%

6 months and 2 years, 3 died and the others recovered completely. Of the 23 patients over 2 years of age 3 died and 20 recovered, 2 with neurological sequelae.

Duration of illness before admission to hospital. Deaths were confined almost entirely to children who had been

TABLE XV. INFLUENCE OF DURATION OF DISEASE BEFORE COMMENCEMENT OF TREATMENT ON OUTCOME OF MENINGOCOCCAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
2 days or less	18 (69%)	2	6 (23%)	26
3 - 7 days	15	1	—	16
More than 7 days ..	4	—	1	5
	37	3	7	47

ill for less than 48 hours before admission to hospital (Table XV). Excluding patients who died within 6 hours of admission, there were only 2 deaths among the 43 children receiving treatment.

Neurological manifestations. Of the 16 patients who showed clouding of consciousness on admission to hospital 5 died, whereas of the 33 who were admitted conscious

TABLE XVI. INFLUENCE OF NEUROLOGICAL MANIFESTATIONS ON OUTCOME OF MENINGOCOCCAL MENINGITIS

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*
Comatose on admission	1	1	2	4
Stuporose on admission	9	—	3	12
Conscious on admission	28	3	2	33
Focal neurological signs	1	—	1	2
Convulsions + clouding of consciousness ..	3 } 5	— } 1	2 } 2	5 } 8
Convulsions + full consciousness	2	1	—	3
No convulsions	33	3	5	41

only 2 died; there were 8 patients with convulsions and of these 2 died (Table XVI). Neurological damage occurred in 4 patients; 1 developed nerve deafness and mental retardation, 2 hydrocephalus and 1 severe disturbance of personality.

Other prognostic criteria. Purpura was noted in 7 patients (13%), 2 of whom died. All had been ill for less than 2 days before admission. Fever subsided within 10 days in 30 (71%) of the patients who survived; 28 of these recovered completely. Only 5 children required hospitalization for more than 28 days and of these 3 had neurological damage.

Discussion

In this series meningococcal meningitis occurred slightly less frequently than pneumococcal meningitis. Excluding the 5 patients who died within 6 hours of admission to hospital, the fatality rate was 3·8%, which is similar to that found in other series.[6,34,44] In contrast to other observers, our mortality was not affected by the age of the patient.[44,45]

As in pneumococcal and influenzal meningitis, clouding of consciousness was the most outstandingly adverse prognostic sign. As almost all the deaths occurred in the group which had been ill for less than 2 days before admission to hospital, the presence of fulminating septicaemia was likely[44,46] and may have led to the fatal outcome in these cases. Complications are rare in meningococcal meningitis,[6,14,44,45] and this was substantiated in this survey. The incidence of neurological damage did not appear to be related to the severity of the illness.

Sulphadiazine remains the drug of choice in meningococcal meningitis.[14,35,47-49] It crosses the blood barrier easily. A CSF level of 7 - 10 mg. per 100 ml. is necessary to obtain effective bacteriostasis, and this can be achieved with doses of 2 - 2½ grains per lb. bw per day.[14,47,49] Theoretically, in order to obtain a high blood level of sulphonamide as quickly as possible it is advisable to administer an initial dose intravenously.[37,49] However, in our series the initial dose was administered IM in most cases, and the results have been satisfactory. Provided the patient is not vomiting, subsequent doses may be given orally or by stomach tube.

It has been found in this series that if a minimal fluid intake of 2½ fl. oz. per lb. bw per 24 hours is maintained no renal complications occur, and the additional use of intravenous or oral alkalinizing solutions[49] appears to be unnecessary.

In view of the excellent results obtained with sulphadiazine there seems to be no need to resort to treatment with broad-spectrum antibiotics except in the rare case which fails to respond to sulphonamides. Penicillin, 250,000 u IM 6-hourly, however, should be given in addition to sulphadiazine to combat any septicaemia.[37,48] Being bactericidal, penicillin is preferable for this purpose to sulphadiazine, which is only bacteriostatic.

MENINGITIS CAUSED BY OTHER ORGANISMS

Salmonella meningitis occurred in 4 patients under 6 months of age; 3 died, and 1 recovered completely. *B. coli* was isolated from the CSF of 1 patient and a non-haemolytic streptococcus from the CSF of another; both these children recovered completely.

PURULENT MENINGITIS OF UNKNOWN ORIGIN

The largest individual group in the survey was formed by patients in whom no organism was found in the CSF microscopically or by culture. This may be due to the present day tendency to use antibiotics indiscriminately for apparently minor illnesses, and an increasing number of patients with meningitis are admitted after a varying period of chemotherapeutic or antibiotic treatment at home.[34] Using the diagnostic criteria laid down by Anglin[50] and Wallgren[51] for the diagnosis of aseptic meningitis,

the 63 cases included in this series were considered to be bacterial in origin. Tuberculous menigitis was carefully excluded.

The age incidence of the patients in this group ranged from 6 days to 8 years; 22 (35%) were under 6 months, 19 (30%) between 6 months and 1 year, and 22 (35%) over 1 year (Table XVIII). There were 33 boys and 30 girls.

Six children died before treatment could be started. The remaining 57 patients fell into 3 treatment groups (Table XVII), viz. (1) 12 patients who received IM penicillin in doses ranging from 0·25 mega-unit 6-hourly to

TABLE XVII. MODE OF THERAPY AND RECOVERY RATE IN MENINGITIS OF UNKNOWN AETIOLOGY

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*	*Refused hospital treatment*
Group I (penicillin + sulphadiazine) ..	5 (42%)	2 (17%)	4 (33%)	12	1
Group II (penicillin + sulphadiazine + streptomycin)	15 (60%)	6 (24%)	3 (12%)	25	1
Group III (penicillin + sulphadiazine + chloramphenicol) ..	12 (60%)	4 (20%)	4 (20%)	20	—

1 mega-unit hourly as well as oral sulphadiazine (2 gr. per lb. bw per day); (2) 25 patients who received IM streptomycin (20 mg. per lb. bw per day) as well as penicillin and sulphadiazine; and (3) 20 patients who received oral chloramphenicol ester in doses varying from 30 to 100 mg. per lb. bw per day as well as penicillin and sulphadiazine (Table XVII).

The clinical picture in these patients was not uniform, because most of them were probably suffering from one of the commoner types of meningitis, viz. pneumococcal, influenzal or meningococcal, each of which has some clinical characteristics of its own.

Results

Of the 63 patients 44 (69%) survived but 12 of these remained with neurological sequelae. This appears in Table XVIII, which also shows the difference in these respects in the three age-groups. The nature of the neurological lesions was similar to that found in the meningitides

TABLE XVIII. INFLUENCE OF AGE ON OUTCOME OF MENINGITIS OF UNKNOWN ORIGIN

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*	*Refused hospital treatment*
Less than 6 months ..	11 (50%)	3 (9%)	8 (36%)	22 (35%*)	—
6 months - 1 year ..	7 (57%)	5 (26%)	5 (26%)	19 (30%*)	2 (11%)
Over 12 months ..	14 (64%)	4 (23%)	4 (23%)	22 (35%*)	—
	32 (50%)	12 (19%)	17 (27%)	63 (100%)	2 (4%)

* Percentage of total cases.

mentioned earlier. No subdural effusions were found. As with the other meningitides all deaths occurred soon after admission to hospital, only 4 of the 17 fatal cases surviving for more than 72 hours.

Factors influencing Prognosis

Clouding of consciousness was the most outstanding factor which affected the prognosis adversely. Of the 29 patients (46%) with clouding of consciousness on arrival in hospital 11 (38%) died and 5 (18%) suffered neurological sequelae, whereas of the 34 patients (54%) who were conscious 6 died (18%) and 7 (21%) were left with neurological damage; the outcome was not affected by

TABLE XIX. INFLUENCE OF NEUROLOGICAL MANIFESTATIONS ON OUTCOME OF MENINGITIS OF UNKNOWN AETIOLOGY

	Full recovery	*Recovery with sequelae*	*Deaths*	*Total*	*Refused hospital treatment*
Comatose on admission 4	12 (41%)	3	6	13	29(46%*) —
Stuporose on admission 8		2	5	16	1 (2%*)
Conscious on admission	20 (59%)	7 (21%)	6 (18%)	34 (54%*)	1 (2%*)
Focal neurological signs	3	3	3	9	
Convulsions + clouding of consciousness 6		4	7	18	—
Convulsions + full consciousness 6	12	2	1	9	1
No convulsions	20	6	9	36	1

* Percentage of total cases.

convulsions provided the patients regained consciousness between seizures (Table XIX).

A bad result was common in patients suffering from malnutrition or diarrhoeal disorders. Advanced malnutrition was present in 8 children; of these 3 died and 4 survived with neurological sequelae. Of the 5 patients with gastro-enteritis 3 died and 2 had neurological residua. There were 17 patients who were known to have received some antibiotic therapy before arrival in hospital; 9 of them had been treated for more than 7 days. A long illness, both with and without treatment, before admission, or fever persisting for more than 10 days after commencement of hospital therapy, had an adverse effect on the outcome.

As in pneumococcal and influenzal meningitis, the prognosis was better in children over 1 year of age. The initial CSF cell-count or sugar level had no detectable prognostic significance.

Discussion

Despite the fact that purulent meningitis of unknown aetiology is becoming increasingly frequent,[36,37] therapy appears to have aroused very little interest. As stated above, most of the patients have probably received inadequate treatment for meningococcal, influenzal or pneumococcal meningitis. Our investigations have shown that the last two diseases are not confined to young infants and that they occur almost as frequently in older children. For this reason best results should be obtained with a combination of drugs effective against *Diplococcus pneumoniae, Haemophilus influenzae* and *Neisseria meningitidis*.

In our series a fairly extensive trial was given to two combinations of drugs. However results in group II (Table XVII), which was treated with penicillin, sulphadiazine and streptomycin, did not differ significantly from those in group III, treated with penicillin, sulphadiazine and chloramphenicol. In view of the fact that neither penicillin nor chloramphenicol was used in optimum dosage in these two groups, better results might have been expected if patients had been given penicillin, 1 mega-unit IM hourly and an initial IT injection of 10,000 - 20,000 u., in combination with sulphadiazine, 2½ grains per lb. bw per day initially (parenterally), and crystalline chloramphenicol 100 mg. per lb, bw per day.

Chloramphenicol, although it crosses the blood-brain barrier easily and has a wide range of antibacterial activity,[36] should probably not be used as the only drug, because some of the pneumococcal cases in this survey were shown to be resistant to it. Furthermore no extensive trial has been carried out with chloramphenicol alone.[27,28]

The question of mutual antagonism is raised whenever a combination of antibiotics is used.[30,52,53] It has been shown, however, that this danger, if it exists at all, is only of importance when drugs are used in inadequate amounts.[39,54]

We, therefore, feel that the therapy recommended above is the best available at present for purulent meningitides of unknown aetiology.

SUMMARY

1. 204 cases of purulent meningitis treated at Baragwanath Hospital over 3½ years ending 31 July 1958 are reviewed.

2. Pneumococcal meningitis occurred in 54 patients, of whom 33 survived; influenzal meningitis in 32 patients, of whom 28 survived; and meningococcal meningitis in 49 patients, of whom 42 survived.

In 6 patients less common organisms were found in the CSF. In 63 patients, of whom 44 survived, no organism was found.

3. Prognostic criteria in the clinical picture were evaluated.

4. Treatment regimens used were assessed. The following treatment schemes are recommended:

(*a*) Pneumococcal meningitis:
1 mega-unit of penicillin IM hourly, with an initial IT injection of 10,000 - 20,000 u., with or without sulphadiazine, 2½ gr. per lb. bw per day.

(*b*) Influenzal meningitis:
crystilline chloramphenicol, 100 mg. per lb. bw per day either orally or parenterally.

(*c*) Meningococcal meningitis:
sulphadiazine, 2½ gr. per lb. bw per day initially parenterally, and penicillin, 250,000 u. 6-hourly IM.

(*d*) Purulent meningitis of unknown aetiology:
Penicillin, 1 mega-unit IM hourly, with an initial IT injection of 10,000 - 20,000 u.; sulphadiazine, 2½ gr. per lb. bw per day; and crystalline chloramphenicol, 100 mg. per lb. bw per day.

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BREAST ABSCESS

A REVIEW OF 220 CASES

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During 1959, 220 patients suffering from breast abscess were treated in the Casualty Department of Groote Schuur Hospital. Infection of the lactating breast with abscess formation has, in fact, been the commonest infection to be treated in this department.

Breast abscess accounted for 19% of all infections that required incision and drainage during the first 3 months of the year (Table I). Since breast infection is practically limited to women in the 20 – 40 age group, this high incidence is even more significant.

This review of cases is undertaken to evaluate our treatment and to investigate the possibility of preventive measures. Adequate follow-up has unfortunately been incomplete because some patients failed to return after the initial treatment.

TABLE I. INFECTIONS REQUIRING DRAINAGE AND INCISION DURING A PERIOD OF 3 MONTHS*

Neck and face	16·3%
Scalp	·6%
Chest wall	—
Abdominal wall	1·6%
Pilonidal sinus and buttocks	5%
Inguinal region and lower limbs	11·6%
Axilla and arm	8·3%
Hand	17%
Foot	5·6%
Ischiorectal and perianal regions	10%
Bartholin's glands	4%
Scrotum	·6%
Breast	19%

* Of the cases treated, 25 were Europeans, 40 Africans, and 157 Coloured patients. Of the 157 Coloured patients, 2 were males and 2 children. The 2 children were 2 years and 5 days of age respectively.

NUMBER OF PREGNANCIES

It is well known that the highest incidence of breast infection occurs after the first pregnancy.[1] The reasons for this are not clear — it may be due to a greater tendency of the nipple to develop cracks in the primiparous woman, or to incomplete emptying of the breast. In our series, the vast majority of cases were primiparous. It is of interest to note that 6 of our patients were not lactating and 4 others had never been pregnant (Table II).

TABLE II. PARITY OF WOMEN WITH BREAST ABSCESS*

First child	70 cases
Second child	18 cases
Third child or more	46 cases
Following abortion	1 case
During pregnancy	5 cases
Not lactating	6 cases
No previous pregnancies	4 cases

(Maternal history available in only 150 cases)

* One of the patients who was not lactating was 89 years old and another 64 years. In the 89-year-old patient no underlying cause could be found. The 64-year-old patient was a diabetic.

It should be noted that there were 16 cases who had been treated for breast abscesses previously. In 10 of these the same breast was involved. Two patients who had developed abscesses on 3 or more occasions had never been pregnant. In both, haemolytic streptococci were cultured.

ORIGIN OF INFECTION

In trying to trace the origin of the infection we felt that contamination by hospital staphylococci might be important. We, therefore, investigated this aspect of the problem. Accurate information was available in 150 cases. Of these 45 were confined at home — 8 of them by district nurses or visiting nurses from local maternity homes. The remaining 105 cases were confined at maternity homes.

It would appear that this high incidence of infection in patients from the maternity homes may be due to the presence of antibiotic-resistant bacteria in the homes, and that infection could be transmitted to the mother even during her very brief sojourn, often lasting 24 hours or less, in the institution. The problem, however, is not limited to the nursing homes in Cape Town; it probably exists throughout the country because some of our cases came from maternity homes in other parts of the country.

Cultures and Sensitivity

Only 3 organisms were found to be responsible for the infections, namely, *Micrococcus pyogenes aureus*, beta haemolytic streptococcus, and enterococcus. Of these *M. pyogenes aureus* accounted for the vast majority of infections (Table III). The sensitivity of *M. pyogenes aureus* to antibiotics is of interest. Among the cases from the maternity homes the staphylococcus was sensitive to penicillin in only 4. Among those who were confined at home the resistant staphylococcus was found in 25. Five of these patients were attended to by district nurses or by visiting nurses from the maternity homes. Staphylococci resistant to penicillin were also obtained from patients who developed breast abscesses 3 – 4 months after leaving a nursing home. (It should be noted that all the staphylococci cultured were sensitive to 'chloromycetin' and erythromycin.) These findings raise to question of carriers of resistant staphylococci.

TABLE III. INCIDENCE OF CAUSAL ORGANISM

Staphylococcus aureus	119 cases
Haemolytic streptococci	6 cases
Staphylococcus aureus plus haemolytic streptococci	3 cases
Staphylococcus aureus plus enterococci	2 cases

In the first place it would appear that either the mother or the baby may become contaminated by a resistant staphylococcus in hospital and then harbour it for as long as 3 – 4 months. This raises the question whether every patient who is admitted to a hospital becomes a carrier, and if so, for how long? In the second place it would appear that the nursing staff of our hospitals may, indeed, be carriers and, again, it may be asked whether all our nurses working in maternity homes harbour penicillin-resistant staphylococci?

Antibiotic resistance seems to be less of a problem with streptococci. In 6 of our patients the infection was purely by beta haemolytic streptococci and in all of them the organism was sensitive to penicillin. It should be noted that 5 of these women were not lactating. From the above it should be clear that the routine prophylactic administration of penicillin in maternity homes is not justifiable, and that it can serve no useful purpose. Sixty-four of our cases received antibiotics, usually penicillin, before the abscesses were incised. Subsequent culture showed that the responsible organisms were sensitive to the particular antibiotic given in only 7 of these cases. Nevertheless, in patients who had received penicillin for long periods, very little pus was found even though the organisms were insensitive. The patients' breasts were more indurated than those of the others and the period of healing after incision was usually prolonged. It seems clear that the prolonged pre-operative use of penicillin predisposes to loculation, which, in turn, renders adequate drainage difficult; and it would appear that antibiotics should rather not be used except on the day of incision.

There is also some doubt about the value of post-operative antibiotics (Table IV) and, in order to assess their efficiency, we have compared cases who received no antibiotics with a random selection of others who received postoperative broad-spectrum antibiotics such as 'tetrex',

erythromycin and 'chloromycetin'. From this comparison it is evident that antibiotics reduced neither the time of healing nor the number of visits required.

TABLE IV. AVERAGE TIME OF HEALING IN CASES TREATED WITH AND WITHOUT ANTIBIOTICS

Loop drainage with antibiotics	35 days
Loop drainage without antibiotics	28 days
Simple drainage with antibiotics	31 days
Simple drainage without antibiotics	26 days

TIME OF ONSET AND DURATION OF ABSCESS

Most of our patients developed their abscesses within the 1st month of lactation (Table V). In an appreciable number of cases, however, abscess formation was delayed for 2 months or more. In 183 cases the duration of the disease was known. Of these 156 came to incision within 14 days of the onset and, of the remaining 27, incision was delayed for up to 60 days. There were no true chronic pyogenic abscesses nor any cases of tuberculosis of the breast.

TABLE V. ONSET OF SYMPTOMS AFTER CONFINEMENT

Less than 7 days	31 cases
7–30 days	101 cases
30–60 days	20 cases
60 days or more	31 cases
Maximum 2 years (still lactating)	
Minimum 2 days	

In 11 cases re-incision was required after a few days up to several weeks. In 1 patient it was necessary to incise the abscess on 4 occasions. Re-incision was usually necessary for abscesses situated elsewhere, but occasionally it was required for re-formation of the abscess deep to the recently healed scar. Almost all the cases which developed recurrent abscesses received antibiotics after the original incision.

METHODS OF DRAINAGE USED

The methods of drainage employed by us may be divided into 2 groups, namely, simple drainage and loop drainage.

1. *Simple Drainage*

A single incision was made and the wound then drained with an ordinary rubber drain, vaseline gauze ('tullegras'), or gauze impregnated with BIPP. The drains were usually shortened after 48 hours and again at subsequent visits until removed on the 10th or 12th day.

2. *Loop Drains*

An incision was made over the centre of the abscess with a counter incision at its most dependent point. A through-and-through loop of corrugated rubber, half an inch wide, was then inserted and the two ends pinned together to form a loop. After 48 hours the rubber was moved slightly and the track irrigated with eusol to promote drainage, and this was repeated at subsequent visits. The loop was removed when the discharge had ceased or diminished to negligible amounts (as determined by staining of the dressings). The loop is left *in situ* for a period of 10 – 15 days.

The method of drainage did not appear to have a significant effect on the time required for healing. With both methods the average time was 31 days. In large breast abscesses, however, the loop drain proved to be superior, because it allowed better drainage during the first 48 hours.

The average time for healing to occur after incision was 31 days (5 – 116 days). This period of 31 days during which the patient attended the hospital compares unfavourably with the usual 10 days of hospitalization required for more serious surgical ailments such as acute appendicitis. Since a large proportion of our patients were working women, the economic consequences were obviously far-reaching.

The average number of visits amounted to 9 – 11, although there have been wide variations. Patients were required to attend 2 days after incision and again 3 – 4 days later. Thereafter, the visits depended on the progress, and it was usually necessary for the patient to attend about every 5th day until the abscess had healed. In many cases visits had to be at longer intervals because of economic reasons and transportation difficulties.

THE USE OF STILBOESTROL

It is generally believed that stilboestrol significantly shortens the healing time of breast abscesses. This is contrary to our experience. However, stilboestrol does relieve the discomfort and pain by diminishing engorgement of the affected breast especially in its own unaffected segments.

A random selection of 123 of our cases received stilboestrol in dosages of 5 – 15 mg. 3 times a day for 5 – 15 days. The average time of healing in this group was 27 days, while in those who did not receive stilboestrol it was 34 days. This difference is hardly significant.

There may be an aesthetic reason for terminating lactation by the use of stilboestrol. In many of our patients in whom lactation had not been stopped, the unpleasant odour of stagnant milk in the dressings was a very genuine complaint. On the other hand, continuation of breast feeding, particularly in the under-privileged group of patients who have been treated by us, is most important from the point of view of the babies, because of the risk of kwashiorkor and other nutritional diseases.[2] We, therefore, usually advise our patients to continue feeding their babies on the other breast.

THE RÔLE OF THE ANTENATAL CLINIC

Infection reaches the breast tissue either by direct entry from the nipple or from the blood stream.[3] In those cases who developed abscesses as a result of infection by antibiotic-resistant organisms 2 – 3 months after leaving a maternity home, the infection almost certainly occurred *via* the nipple. Other evidence in favour of this being the common route of infection, is the higher incidence of breast abscess in primiparous women who suffer more frequently from cracked nipples. (However, very few of our cases gave a history of a preceding cracked nipple.)

Ninety-one of our patients attended antenatal clinics, but only 27 of these received any nipple care during their pregnancies. The remainder either received no instructions on antenatal care, or ignored the instructions given. It is our impression that improved care of the nipples during pregnancy, which will reduce the incidence of cracked nipples, might well reduce the number of breast abscesses

incurred, especially in the primiparous women who were confined in maternity homes.

SUMMARY

220 cases of breast abscess are reviewed. The effects of antibiotics and stilboestrol on the period of healing are discussed. The rôle of maternity homes and antenatal clinics in relation to these abscesses are briefly discussed.

I should like to thank Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital, Observatory, Cape, for the permission to publish these cases; and Prof. J. H. Louw, Head of the Department of Surgery, and Mr. T. Schrire, surgeon in charge of the Casualty Department, for their interest and encouragement.

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WORKMEN'S COMPENSATION CASES

The Workmen's Compensation Commissioner, who is administering the Accident Fund, is handling trust monies and his actions are subject to Government audit. It must be appreciated that the Commissioner, in accordance with the provisions of the law, can only defray the *reasonable* medical expenses incurred by, or on behalf of, an injured workman.

Fees for the treatment of injured workmen have been laid down in consultation with the Medical Association of South Africa. The fees which provide for a variety of conditions resulting from trauma are considered reasonable and cannot be amended.

The Commissioner, in administering the Workmen's Compensation Act, is anxious to retain the cooperation of the medical profession; hence his reluctance in the past to take action against the few doctors who had acted contrary to accepted principles.

The following suggestions on what to do and what not to do in dealing with workmen's compensation cases, are published for the information and guidance of doctors.

DO'S AND DONT'S FOR DOCTORS

DO:

1. Be accurate in furnishing information.
2. Give a full clinical description of the condition found on the first examination and answer all questions fully on medical forms.
3. Give full details of treatment rendered or procedures carried out.
4. Furnish details of complicating or hindering factors which retard recovery.
5. X-ray examinations yourself only when the services of a radiologist are not reasonably available.
6. Book the workman back to work as soon as this can be done without harm to the patient.
7. Indicate on the final medical report the date on which the workman is fit to resume work.
8. Describe fingers and joints by name and not number.
9. Prescribe only essential drugs in moderate quantities and repeat prescriptions if necessary.
10. Make sure that the contents of medical report forms accurately reflect the facts since the forms are in the nature of a medical certificate.
11. Furnish reports promptly to the employer; to the Commissioner only when requested to do so.

DON'T:

1. Give vague descriptions of injuries such as 'backstrain', 'contact dermatitis', etc.
2. Over-treat, over-visit or over-prescribe.
3. Prescribe expensive medicines where less costly drugs will suffice.
4. Give physiotherapy when there is a physiotherapist available.
5. Claim for a condition not covered by the medical reports.
6. Send the workman back to work on a Monday if he is fit before then.
7. Leave it to the employer to fill in the date on which the workman is fit for work.
8. Let your nurse do any treatment other than changing dressings.
9. Hesitate to consult a specialist where progress is unsatisfactory.
10. Claim for work not done.
11. Delay submission of medical reports and accounts.
12. Claim for visits where there is a fixed fee.

COLLEGE OF PHYSICIANS, SURGEONS AND GYNAECOLOGISTS OF SOUTH AFRICA: KOLLEGE VAN INTERNISTE, CHIRURGE EN GINEKOLOË VAN SUID-AFRIKA

EXAMINATION RESULTS

The following candidates were successful in the examinations of the College of Physicians, Surgeons and Gynaecologists of South Africa, held in Johannesburg in October this year:

Fellowship of the College of Physicians of South Africa

Dr. I. Law, Johannesburg.
Dr. I. N. MacLeod, Durban.

Fellowship of the College of Surgeons of South Africa — Final

Dr. C. J. Mieny, Johannesburg.
Dr. H. F. Pieterse, Bellville, Cape.

Fellowship of the College of Surgeons of South Africa — Primary

Dr. C. G. Apostolis, Johannesburg.
Dr. F. J. Badenhorst, Johannesburg.
Dr. I. Bruk, Sea Point, Cape.
Dr. T. Coetzee, Pietermaritzburg.
Dr. D. Hagen, Roodepoort, Transvaal.
Dr. A. L. Mendelow, Johannesburg.
Dr. J. C. Sandeman, Johannesburg.
Dr. D. Sevel, Johannesburg.
Dr. A. J. Vosloo, Johannesburg.

Diploma of Midwifery of the College of Obstetricians and Gynaecologists of South Africa

Dr. J. E. Berge, Pietermaritzburg.
Dr. D. V. Cohen, Johannesburg.
Dr. A. Rayman, Johannesburg.
Dr. E. M. A. Rothschild, Johannesburg.
Dr. I. L. Safro, Krugersdorp.
Dr. J. D. Walker, Pietermaritzburg.

Fellowship of the Faculty of Anaesthetists of the College of Physicians, Surgeons and Gynaecologists of South Africa — Final

Dr. N. L. Wulfsohn, Johannesburg.

Fellowship of the Faculty of Anaesthetists of the College of Physicians, Surgeons and Gynaecologists of South Africa — Primary

Dr. A. C. Buys, Pretoria.
Dr. B. S. Mullan, Pretoria.
Dr. D. J. Rossouw, Pretoria.
Dr. G. Sher, Pretoria.

Diploma in Psychiatric Medicine of the College of Physicians of South Africa — Final

Dr. B. Braude, Johannesburg.
Dr. C. G. A. Simonsz, Queenstown.
Dr. E. Smit, Krugersdorp, Transvaal.

Diploma in Psychiatric Medicine of the College of Physicians of South Africa — Primary

Dr. G. P. J. van Niekerk, Queenstown.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING OCTOBER 1960

International Social Security Association, Permanent Medico-social Committee, meeting, Geneva, 21 - 23 November 1960. 154 rue de Lausanne, Geneva, Switzerland.

World Health Organization, Scientific Meeting on Rehabilitation in *Leprosy*, Vellore, India, 21 - 29 November 1960. Palais des Nations, Geneva, Switzerland.

International Congresses on Tropical Medicine and Malaria, Conference on Malaria in Africa South of the Sahara, Nairobi, Kenya, November 1960. Dr. M. Pinotti, Departamento Nacional Saude, Ministério Saude, Rio de Janeiro, Brazil.

World Health Organization, Expert Committee on *Specifications for Pharmaceutical Preparations*, Geneva, 5 - 9 December 1960. Palais des Nations, Geneva, Switzerland.

World Health Organization, Inter-regional Conference on Techniques of Surveys on the *Epidemiology of Mental Disorders*, Italy, 6 - 15 December 1960. Palais des Nations, Geneva, Switzerland.

All-Union Conference on Tissue Incompatibility and Transplantation of Organs and Tissues, Novosibirsk, USSR, 1960, Institute of Experimental Biology and Medicine, Siberian Department, Academy of Sciences of the USSR, Novosibirsk, USSR.

World Health Assembly, 14th, New Delhi, 7 February 1961. WHO, Palais des Nations, Geneva, Switzerland.

International Academy of Pathology, 50th Annual Meeting, Chicago, 22 - 29 April 1961. Dr. F. K. Mostofi, Armed Forces Institute of Pathology, Washington 25, D.C., USA.

Twelfth International Hospital Congress, Venice, June 1961. Capt. J. E. Stone, Secretary General, International Hospital Federation, 34 King Street, London, E.C.2, England.

PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

WESTDENE PRODUCTS (PTY.) LTD.

Westdene Products (Pty.) Ltd. Medical Book Department announce that the following new books have been received and are now available on application to this Department at P.O. Box 7710, Johannesburg:

Surgery in the Aged by F. Glenn, S. W. Moore and J. M. Beal	162s. 0d.
Rose and Carless' Manual of Surgery, consulting editor Sir Cecil Wakely	96s. 0d.
Clinical Toxicology by C. J. Polson and R. N. Tattersall	48s. 6d.
Elements of Vital Statistics by B. Benjamin	65s. 3d.
Blood Flow in Arteries by D. A. McDonald	48s. 6d.
Visual Aids in Cardiologic Diagnosis and Treatment, edited by A. M. Master and E. Donoso	85s. 0d.
Medical History Taking by I. Stevenson	60s. 3d.
Principles of Human Genetics by C. Stern	71s 3d.
Outpatient Treatment of Schizophrenia by S. C. Sher and H. R. Davis	49s. 0d.
Mental Retardation, edited by P. W. Bowman and H. V. Mautner	106s. 3d.
Diseases of the Newborn by A. J. Schaffer	168s 0d.
The Health of the Community by F. Brockington	42s. 6d.
Antenatal and Postnatal Care by F. J. Browne and J. C. McC. Brown (1960 edition)	52s. 6d.
Obstetrical and Gynaecological Pathology by R. E. Rewell	57s. 6d.
Hypnotism for Medical and Dental Practitioners by A. A. Mason	36s. 6d.
Electrical Activity of the Nervous System by M. A. B. Brazier (1960 edition)	42s. 6d.

Contrary to the information published in the list of 'Books Received' in the issue of the *Journal* for 5 November, Messrs. Westdene Products are also local agents for all publications of Messrs. C. V. Mosby Company of St. Louis, USA.

QUESTIONS ANSWERED : VRAE BEANTWOORD

CHILBLAINS

Q—*What are the present-day views on the treatment and prevention of chilblains? Are vasodilator drugs likely to help?*

A—Chilblains, the pernio syndrome, perniosis, erythrocyanosis, erythrocyanosis frigida, erythrocyanosis crurum puellarum, nodular vasculitis, erythema induratum, and Bazin's disease are some of the names applied to different stages of the same disease entity. The use of the single term 'chilblains' to embrace all of these makes for simplicity and clarity.

The underlying cause of chilblains is low-grade cold and damp, producing vasospasm in individuals whose cutaneous vessels are peculiarly susceptible. In the acute stage the lesion presents as an intensely itchy, swollen, erythematous blotching of the skin of the fingers and toes and occurs commonly in young female adults. Induration and subcutaneous nodules over the calves, which may progress to painful and indolent ulceration, characterize the chronic stages.

Prevention

'Prevention is better than cure' is literally true in the management of this homely condition. Adequate warm clothing, warm gloves and stockings, well-fitting comfortable footwear, and regular exercise are mundane measures well worth conscientious application, though not all of these are compatible with modern concepts of fashionable dress — short skirts, sheer stockings, and elegant shoes.

Too rapid warming of the extremities either before a heater or open fire, or in hot water, on coming indoors after exposure to cold, may precipitate the lesion.

In the early stages chilblains are seasonal (appearing with cold weather and disappearing with warm weather) and reversible, and complete spontaneous recovery may occur after the age of 20 years, while girls may cease to suffer from attacks after marriage. In the chronic stage, however, irreversible tissue damage will have occurred.

Treatment

Definitive treatment is largely empirical. Calcium gluconate therapy, two 7½ gr. tabs *q.d.s.*, appears to be of benefit, and thyroid extract, ½ - 1 gr. daily for 2 - 4 weeks, is worthy of trial. Both these drugs should be commenced at the onset of the cold season. Local applications are legion: painting with tincture of iodine, and applications of icthyol, menthol, zinc cream with 2% tar, and salicylate ointments are as useful as any others. Vasodilator drugs would seem to be rational therapy, but the response is far from convincing either in the acute or the chronic phase.

For the severe chronic lesions with marked induration and persistent ulceration involving the skin of the calves, lumbar sympathectomy should be seriously considered to relieve the chronic arteriolar vasospasm. This will give subjective improvement of the sensation of coldness, burning, and heaviness of the limb and lead to healing of the ulcers, but the induration will be unchanged and scaly atrophic areas will remain to mark the sites of healed ulceration.

Readers are invited to submit questions for this column to the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town.

PASSING EVENTS : IN DIE VERBYGAAN

Iced Water for Burns. In a recent edition of the *Journal of the American Medical Association*, a physician recommends immersion in iced water as first aid for burns covering up to 20% of the body. 'In every patient thus treated, gratifying relief was expressed at once', says Dr. Alex G. Shulman, of Los Angeles. His method is to place the burned area immediately into a basin of water, ice cubes and disinfectant. For burns on parts of the body where immersion is impractical, Dr. Shulman recommended application of towels chilled in a bucket of iced water. He said he had treated 150 patients in this manner. He called the iced-water treatment 'far more effective first-aid treatment than the usual first-aid measure of applying butter or grease, which will only have to be painfully removed later by the attending physician'.

* * *

Dr. M. M. Suzman, of Johannesburg, left for overseas on 11 November to attend an International Symposium on Long-term Anticoagulants which was held in London on 18 - 19 November. Dr. Suzman was invited to participate in this symposium, which was under the Chairmanship of Sir George Pickering and was sponsored by Evans Medical Ltd., and delivered a paper entitled 'An evaluation of the effect of long-term anticoagulant therapy on the prognosis of coronary-artery disease'. Dr. Suzman will return to the Union at the end of January 1961.

* * *

Mr. Cecil Morris, orthopaedic surgeon, of Johannesburg, has recently returned from the USA, where he visited a number of orthopaedic centres.

* * *

University of the Witwatersrand, Medical Graduates Association. The closing date for the forthcoming postgraduate course, arranged by the Medical Graduates Association and the Faculty of Medicine of the University of the Witwatersrand, to be held on 16 - 21 January 1961, will be 15 December. Members who intend applying to participate in this course are urged to do so as soon as possible.

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Medico-Surgical Cinema. The annual prize for Medico-Surgical Cinema, which is awarded by *La Presse Médicale*, and valued at 1,000 NF cash (which, if needed, can be divided) and various other awards, will be given during the last session of the course of *Actualités Médico Chirurgicales* at the *Nouvelle Faculté de Médecine de Paris* on 14 March 1961.

The jury will consider the didactic value of the film as well as its cinegraphic quality. Only 16 mm. size films will be admitted. The time of projection must not exceed 30 minutes.

Films are to be sent to the *Secrétariat du Journal La Presse Médicale*, 120 Boulevard Saint-Germain, Paris VI°, before 1 February 1961.

Prizes will be awarded to the compilers of the best films. All films may be exhibited including those subsidized or produced by a laboratory or a firm.

* * *

Dr. M. P. (Lowna) Keet, M.Med. (Paed.) (Stellenbosch), het as kinderspesialis begin praktiseer te Bellville Mediese Sentrum, Volkskasgebou, Bellville. Telefone: Spreekkamer 97-3926, woning 97-3321.

Dr. M. P. (Lowna) Keet, M.Med. (Paed.) (Stellenbosch), has commenced practice as a paediatrician at Bellville Medical Centre, Volkskas Building, Bellville. Telephones: Rooms 97-3926, residence 97-3321.

* * *

Princess Alice Orthopaedic Hospital, Opening of Orthopaedic Out-patients' Department. As from 1 November 1960 all new, cold or long-term orthopaedic out-patient clinic cases which would hitherto have been referred to the Groote Schuur Hospital Orthopaedic Department, should now be referred to the Princess Alice Orthopaedic Hospital, Retreat, Cape. Since these clinics will be on a strict appointment basis, doctors are asked, in referring any such case, to write to the Clinical Secretary, Princess Alice Orthopaedic Hospital, Retreat, requesting such an appointment, or in more urgent cases obtain an appointment by telephoning 71-6661. However, all fracture and traumatic cases will continue to be referred to the Orthopaedic Department, Groote Schuur Hospital.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 28 November at 5.10 p.m. in the Institute Lecture Theatre. Prof. P. Tobias, Professor of Anatomy, University of the Witwatersrand, will speak on 'New thoughts on the evolution of human races'.

* * *

Johnson and Johnson Awards for Postgraduate Clinical Study in South Africa. The following medical practitioners have each been granted one of these awards: Dr. G. G. Airey, Umtata; Dr. P. D. Beck, Port Elizabeth; Dr. P. A. Goosen, Vrede; Dr. J. A. Meyer, Bethal; and Dr. I. Sagor, Worcester.

Annual Inter-university Surgical Symposium. This symposium will be held on Thursday 24 November at 8.15 p.m. in the Physiology Lecture Theatre, Medical School, University of Cape Town, Observatory, Cape. The speakers are: (1) Prof. D. J. du Plessis (University of the Witwatersrand) 'A study of sialography in parotid swelling'; (2) Prof. C. H. Derksen (University of Pretoria) 'Karsinoom van die mamma'; and (3) Prof. J. H. Louw (University of Cape Town) 'Selection of patients for aorto-iliac surgery'. All practitioners and medical students are cordially invited to attend this symposium.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

ANTHICAL CREAM

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of Anthical cream as a companion product to Anthical lotion, and supply the following information:

Anthical cream contains mepyramine maleate 1·5% w/w and zinc oxide 15% w/w in a vanishing-cream type base; colouring has been added to impart a flesh-pink colour to the product.

Anthical cream provides an alternative to the lotion when an emollient preparation is preferred. Both preparations have similar indications, viz. for the relief of sunburn, itching, nettle stings and prickly heat, the emphasis being placed on the use of the cream in sunburn.

Anthical cream should be rubbed gently into the affected parts. It may be applied 3 times daily. Like other topical antihistamine preparations, Anthical cream (and the lotion) should be used with care on extensive broken and eczematous skin surfaces.

Further information about Anthical cream is obtainable from the Medical Department, Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

ACTRIOL

Keatings Pharmaceuticals Ltd. announce the introduction of Actriol, a new Organon product, and supply the following information:

Actriol cream contains 2·5 mg. per g. of 16-epi-oestriol and is used for the treatment of acne vulgaris.

Actriol atrophies and suppresses the activity of the sebaceous glands without producing any of the usual oestrogenic side-effects.

The clinical effectiveness of Actriol has been proved by the local application of only 1 mg. per g.; after about 2 months treatment in both sexes 73% of cases showed great improvement.[1]

The affected area is washed with warm water and dried.

Actriol is then rubbed in twice daily. Half an inch of cream is usually sufficient for one application.

Actriol, manufactured by Organon Laboratories Ltd., is available in 15 g. tubes.

Further information is available from Keatings Pharmaceuticals Ltd., P.O. Box 256, Johannesburg.

1. Peterkin, G. A. G. (1959): A.M.A. Arch. Derm., **80**, 1.

STAPHCILLIN

B.L. Pharmaceuticals (Pty.) Ltd., announce the introduction of Staphcillin, a new synthetic penicillin, and supply the following information:

A new synthetic penicillin capable of destroying the resistant staphylococcus germ has been produced by Bristol Laboratories and a report to the medical profession was made on 7 September at a symposium sponsored by the State University of New York, Upstate Medical Centre, at Syracuse, New York.

The chemotherapy of staphylococcal infections is complicated by the variable character of the species. It has been one of the most remarkable features of penicillin that it could control staphylococcal infections. However, it was already known in the beginning of the penicillin era, approximately 15 years ago, that a certain percentage of the pathogenic strains of *Staphylococcus aureus* showed resistance to penicillin and produced a penicillin-destroying enzyme, penicillinase.

Barber in Great Britain (1948) was the first to draw attention to a general rise in resistance of the staphylococci to penicillin. She found that of 100 cases of staphylococcal infection in the Postgraduate Hospital, London, in 1948, 59 were penicillinase-producing strains, resistant to penicillin G, whereas in 1946 only 14·1% of the strains were resistant. Since then hundreds of publications have appeared on this subject. In general it can be said that the extensive use of penicillin G, particularly in hospitals, has resulted in a complete change in the staphylococcal flora. It is assumed that penicillin G has destroyed most of the staphylococcal strains sensitive to this antibiotic and that the resistant, penicillinase-producing strains have taken over. Some hospitals report that percentages up to 90% of these staphylococci, isolated from patients or from the carriers among their staff personnel, are resistant to penicillin G.

From the therapeutic point of view, the situation has not been altogether gloomy and hopeless, but it has been far from happy. Various antibiotics — and combinations of antibiotics — have been employed with some, if hardly an adequate, degree of success. Staphylococci have not invariably resisted every therapeutic effort. But the proportion of staphylococcal strains resistant to each drug or combination varies from community to community and even from hospital to hospital, with the variation ranging from as little as 20% to as much as 90% resistance. Mortality has been high from certain types of staphylococcal infections — those accompanied by staphylococcal septicaemia, endocarditis, pneumonia or meningitis. Among the pneumonias, those occurring in infancy and those complicating influenza have been particularly deadly. The need for some new therapeutic agent — one specifically active against resistant staphylococci and with a high margin of safety for the patient — has been acute.

Until the synthetic breakthrough of a year ago, when scientists of Bristol Laboratories in collaboration with scientists of Beecham Laboratories developed the first synthetic penicillin, alpha-phenoxyethyl penicillin (Bendralan), the idea that such a drug could be found in a penicillin seemed most unlikely.

Now, actually less than 12 months after the announcement of alpha-phenoxyethyl penicillin, a new development has been reported: the second synthetic penicillin, a result of co-operative research in the synthetic penicillin field by American and British scientists. Chemically the new product is 2, 6-dimethoxyphenyl penicillin. It will be made available to the medical profession under the name Staphcillin.

This new synthetic penicillin is just what Dr. Ernst Chain, Nobel prize winner and early pioneer in penicillin development in the 40's, wanted when he emphasized the staphylococcal problem at a symposium in October, 1959, in Syracuse. What is needed, he stated, is a penicillin that is both antibacterial and anti-enzymatic; that is, not destroyed by penicillinase.

Staphcillin proved to be insensitive to destruction by the enzyme penicillinase. *In vitro* it generally shows less activity than penicillin G, but against resistant staphylococci, it gives an exciting performance. All strains of staphylococcus are uniformly sensitive to the new penicillin, regardless of how much resistance they have to penicillin G. In pharmacologic and toxicologic studies the new compound was found to have the same order of toxicity as penicillin G; that is, it is practically non-toxic. It is also practically devoid of irritant liability, being less painful than penicillin G when injected.

In the clinical trials to date the drug has lived up to expectations. Nearly 90% of patients with resistant staphylococcal infections either have been completely cured or considerably improved by Staphcillin.

Staphcillin represents a breakthrough into the resistant area where adequate therapy has been lacking. It promises to be a major step forward in bringing the staphylococcus under the control of Man.

Staphcillin is available in sterile, dry-fill vials containing 1·0 g. of the monohydrate of sodium dimethoxyphenyl penicillin. The product has to be reconstituted with 1·5 ml. sterile distilled water to give a total finished volume of 2·0 ml.

Full details are available from B.L. Pharmaceuticals (Pty.) Ltd., P.O. Box 2515, Johannesburg.

DILODERM

Schering Corporation USA introduce the first chlorinated steroid, Diloderm (dichlorisone acetate), which was developed to provide superior topical steroid effectiveness, and supply the following information:

With Diloderm, anti-inflammatory activity is markedly enhanced at the local level without a corresponding increase in metabolic effects on systemic administration.

Totally different from all present steroids, Diloderm is chlorinated — the first such corticoid to be made available for clinical application. This unique structure may account for the unique specificity of Diloderm for topical use on the skin. Diloderm provides increased topical benefits in allergic, inflammatory, and pruritic skin disorders responsive to topical steroid therapy including: atopic dermatitis, allergic dermatitis, infantile eczema, nummular eczema, eczematoid dermatitis, contact dermatitis, pruritus ani and vulvae, pruritus with lichenification, and disseminated neurodermatitis. Intramuscular administration of 25 - 80 mg. daily in humans confirms animal studies and demonstrates that Diloderm produces no changes in serum levels of sodium, phosphorus, calcium, magnesium, and alkaline phosphatase or in circulating eosinophils, 17-ketosteroid excretion, potassium balance, and nitrogen balance.

In contrast to the metabolic effects associated with other active anti-inflammatory steroids, the relative absence of such effects in therapy with Diloderm has been further demonstrated by oral administration of Diloderm to patients undergoing therapy with conventional systemic steroids. In such cases, patients relapsed with oral dosages of Diloderm of 60, 80, 120, and 200 mg. daily. One patient, maintained on 10 mg. of prednisone daily, had a prompt relapse when given Diloderm in oral dosages of up to 240 mg. daily.

The general absence of absorption after topical application of Diloderm is shown by lack of systemic effect reported by investigators. In addition, eosinophil counts performed at 4 and 24 hours showed no significant change in subjects on whom 20 g. of Diloderm Cream was applied to the body and thoroughly massaged in.

Diloderm is supplied as Diloderm Cream 0·25% in 5 g. tubes and as Neo-Diloderm Cream 0·25% in 5 g. tubes.

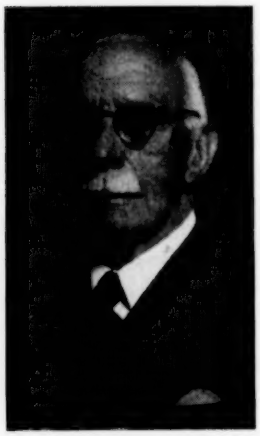
Further information may be obtained from Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

IN MEMORIAM

ALEXANDER SIMPSON WELLS, M.A., M.D. (GLASG.), F.R.C.S. (EDIN.)

Dr. T. Shadick Higgins, of Cape Town, writes:

The death of Dr. A. Simpson Wells, which took place suddenly on 8 September 1960, severs one of the remaining links with medicine as practised at the Cape at the beginning of the present century.



Dr. Simpson Wells

He had reached the age of 85 years. Born in Glasgow on 3 September 1875, he was the son of James Wells, D.D., Presbyterian minister, and great-nephew of Sir J. Y. Simpson, pioneer of chloroform anaesthesia. In 1904 he married Edith Boyd Henderson, of Aberdeen, Scotland, by whom he is survived.

He was educated at the Glasgow High School and the Glasgow Academy, and graduated at the University of Glasgow, afterwards studying at Berlin and London. He held resident posts in the Glasgow Western Infirmary (under Sir Hector Cameron, who had been house surgeon to Lister), the Glasgow Royal Infirmary, and the Royal Maternity Hospital, Glasgow.

Simpson Wells saw service in South Africa in three wars. He left Britain in 1900 as civil surgeon in the British Forces in the Anglo-Boer War, throughout which he served, first with the 4th Royal Lancashire Regiment and later with the 1st Mounted Infantry. In World War I he served in the South West African campaign, mainly at Okahandja. In World War II he worked at the Wynberg Military Hospital and then as Medical Officer in charge, Royal Air Force training camp, Heany, Southern Rhodesia.

After the Anglo-Boer War he undertook plague duty at Cape Town under the late Dr. J. A. Mitchell, and then, after a visit to Scotland, engaged himself as assistant to the late Dr. Robert Sharp at Woodstock. In 1903 he started his own practice in central Cape Town, which he continued for over 45 years, at Orange Street, New Church Street, and Kloof Nek Road successively, retiring about 1949. In the last 10 years of this long period he was in partnership first with Dr. Robert McDonald and then with Dr. Charles Saunders. Though Simpson Wells was always a general practitioner, he had a special interest in obstetrics and gynaecology, which he practised extensively.

In 1917 he began a lifelong association with St Monica's Maternity Home for non-Europeans, of which he was honorary medical superintendent for many years, and took an active part in the training of Coloured midwives and in the conduct of the first antenatal clinic to be set up in Cape Town. In 1918 he was appointed (with Dr. Sutton Charnock) as senior medical officer of the Peninsula Maternity Home, and in the following year as clinical lecturer and examiner in Obstetrics, University of Cape Town, a post which he held for about 30 years. He was honorary medical officer of the Child Life Protection Society at the time of the founding of the Lady Buxton Home and took a great interest in the training of mothercraft nurses. He also served as committee member for the King Edward VII Order of Nurses, the Service Dining Rooms, the Institute of Race Relations, and the National Cancer Association.

In the Medical Association of South Africa Simpson Wells played a long and honourable part. He was a prominent member of the Cape Western Branch of the British Medical Association from the days before the fusion which resulted in the formation of the South African Medical Association, and he was Branch President in 1919. He took a leading part in the acquisition on behalf of the Branch of the premises at 35 Wale Street, Cape Town, which were converted into the pleasant board room and library that for long housed the Branch, and where the present building, in which the Head Office of the Association is situated, was later erected by the Association and the Branch jointly.

Simpson Wells was a man of strong Christian principles, and a lifelong member of the Presbyterian Church. He was a teetotaller by conviction and a student of the problems of alcoholism. His only hobby, apart from walking, he found in keeping abreast of medical advances. He made a lasting mark in the history of medicine in the city and country of his adoption, and his death leaves a gap that will not easily be filled. His memory will long be treasured by a vast number of friends, colleagues, patients and students. He is survived by his widow, a son and two daughters; his elder son was killed in a climbing accident on Table Mountain in 1931.

ANDREW CAMPBELL WATT, M.B., CH.B. (EDIN.) M.R.C.P. (EDIN.)

Drs. A. V. Bird, J. C. Gilroy and S. Jacobson, of Johannesburg write:

At midnight on 28 October Dr. Andrew Watt was killed in a motor accident while on his way to visit a patient in hospital. Not only has the medical profession in South Africa lost an outstanding and talented figure, but many people, both here and in Britain, have lost a true and esteemed friend.

Dr. Watt

Dr. Watt was 45 when he died. He was born in Germiston and was the son of Dr. Andrew Hutton Watt, who came to this country just after the war of 1900, and was one of the founders of the Chamber of Mines Hospital at Cottesloe.

Having been educated at St. John's College and at Haileybury, Andrew studied medicine at Edinburgh University where his father had held the post of assistant to Sir Harold Stiles before he emigrated to South Africa.

During his university days Dr. Watt held the Conan Doyle Scholarship, and when he qualified he decided to take up neurosurgery as his career. He became an assistant to Mr. Norman Dott in the Department of Neurosurgery of Edinburgh University and, shortly before the invasion of Normandy, he joined the late Prof. Sir Hugh Cairns in Oxford.

Just after the outbreak of war in 1939 the Royal Army Medical Corps established at St. Hugh's College, Oxford, a hospital for treating head injuries and neurological diseases, and Dr. Watt became a member of the staff of this hospital.

When the invasion of Europe took place he landed in Normandy on D+1 day as the neurologist to a mobile neurosurgical team, and after the war he returned to the Hospital for Head Injuries which had now expanded and moved out to Wheatley.

In 1948 he returned to South Africa and became a lecturer at the University of the Witwatersrand and neurologist to the Johannesburg General Hospital. He worked as a member of Mr. R. Krynauw's neurosurgical unit and, on Mr. Krynauw's retirement, he played a prominent part in keeping the neurosurgical unit intact during a difficult period.

In spite of his hospital activities and a very busy consulting practice he found time to visit the Baragwanath and Coronation non-European hospitals once a week until a full-time neurosurgical department was established to provide a service for these patients.

On his mother's side Andrew came of a long line of lawyers, so that it was very natural that he took great interest

in the forensic side of his work as a neurologist. In the law courts he quickly established a reputation as an expert witness and his opinion was widely sought after by his legal colleagues. He appeared in many famous law cases during the past 10 years and was a founder member of the Medico-legal Society of Johannesburg. He made original contributions to medical literature on head injuries, hallucinations, and alcoholism.

Apart from his academic and professional attainments Andrew had a warm humanity and a generous nature that endeared him to those of us who have known him and worked with him. Anyone in trouble could always depend on a sympathetic and practical approach to his difficulties and no-one who asked for his help ever came away without it.

He was a man of profound culture, widely read, and with an unusual command of the English language.

On occasions his manner was gruff and abrupt, but he had a Puckish sense of humour, and nothing gave him more pleasure than deflating pomposity. However, this was done in a manner which few of his victims resented for any length of time.

His untimely death leaves an enormous gap in the ranks of the medical profession of South Africa which will not easily be filled.

His widow and son and daughter survive him.

Dr. A. L. Agranat, of Johannesburg, writes:

Doctors in Johannesburg were shocked to learn of Andrew Watt's death which followed a car accident during the night of 27 October. It was in response to a call about midnight to see a patient of his at a nursing home that he met with this sad end. His many friends were dumbfounded and grief-stricken at the incredible news.

Andrew was the son of a well-known Johannesburg doctor, the late Andrew Hutton Watt, whose reputation had become a byword in the medical world. To have grown up in the atmosphere of such admirable traditions accounted for many of the high principles by which Andrew lived.

His somewhat gruff exterior may have been forbidding until the real Andrew Watt was revealed. He was incapable of an unkind thought, far less an unkind action. He seemed to sense when his help and friendship were needed, and he never failed to give generously of that unfailing store. There was nothing soft about Andrew and emotional display was foreign to him. His sincerity penetrated the atmosphere whether in convivial surroundings or in his professional life even if differences of opinion existed.

Andrew Watt's reputation as a neurologist was established in Southern Africa and many reports reached us about the high esteem in which his name was held by some of the leading neurologists in Britain. He qualified at Edinburgh University in 1938 and obtained the M.R.C.P. degree at Edinburgh University in 1947. He spent several years working in Edinburgh with Mr. Norman Dott and subsequently worked under Sir Hugh Cairns at Oxford. He served in the British Army during the last war in Belgium and in France in the capacity of Specialist Neurologist with the rank of Major.

He returned to South Africa in 1948 and very soon established a reputation as one of the leading neurologists. He developed a large practice and, with the two colleagues who joined him later as partners, enjoyed a happy association which existed up to the time of his tragic death. He was an expert medical witness and his services were frequently called upon in important legal issues — work which he loved because he was at his best in a verbal battle of wits.

It is almost impossible to grasp that he has gone. How we shall miss that lovable character, his unostentatious sincerity, his comradely bantering — and his outstanding brain. To his friends he always gave far more than he took. His honesty of purpose and selfless devotion made Andrew a unique personality and his passing has left both his friends and his profession bereft.

ALEC REICHLIN, M.B., CH.B. (LEEDS), D.PHYS. MED. (RAND)

Prof. N. Sapeika, of Cape Town, writes:

The sudden tragic death of Dr. A. Reichlin a few minutes after he was struck by a car has come as a great shock to his many friends.

Alec was born in Palestine 61 years ago, when that country was under Turkish rule, but he spent his early years in South Africa before proceeding to Leeds University where he graduated in medicine in 1928. He studied under Lord Moynihan, and with strong encouragement from that famous surgeon he would have specialized in surgery had circumstances permitted.

Dr. Reichlin

He entered general practice at Postmasburg near the Kalahari border in 1930, and remained there, apart from the war years, until 1948. In the course of his work he performed surgical operations, many of major or emergency nature, and also obstetric procedures. These were done under most difficult conditions; for instance there was no laid-on water or electricity. He also established a hospital to meet the needs of the population between Kimberley and Kuruman. In those days it took a whole day to travel by car (and ferry across the Orange River) from Postmasburg to Kimberley, a distance of 150 miles. He also served as railway doctor and medical officer of health, and attended to the workers of three manganese mines, for whom he instituted proper feeding and housing schemes.

During World War II Alec served in North Africa as a captain in the Medical Corps, attached to the Kaffrarian Rifles. He would have been captured at Tobruk had he not been called back a short while before to duties in the Postmasburg hospital. After the war he was in private practice for a period before qualifying for the Diploma in Physical Medicine in 1948, later becoming head of the Physiotherapy Department at Groote Schuur Hospital. He was a member of the Medical Association of South Africa for 32 years, from the time he qualified as a doctor.

In the course of his lifetime he aided a multitude of people in his quiet, efficient manner. Even in recent years when he suffered from ill-health he performed his duties with a kindly disposition. His interests were not confined to medicine, but included music, art and literature, and he was regularly to be seen at concerts, exhibitions and lectures.

The unfortunate accident occurred a few days before he was due to leave for a visit to the land of his birth, in which he showed active interest since his student days. His colleagues and friends extend to his wife and two children their deepest and heartfelt sympathy.

CORRESPONDENCE : BRIEWERUBRIEK

PATHOGENESIS AND SURGICAL MANAGEMENT OF PEPTIC ULCER

To the Editor: Mr. A. Dickson Wright, F.R.C.S., on his return from South Africa, has kindly drawn my attention to a paper[1] by Professor du Plessis which appeared in the issue of the *Journal* for 6 February entitled 'Some aspects of the pathogenesis and surgical management of peptic ulcer'.

I have read it with particular interest because the author suggests that through failure of the pyloric sphincter, reflux of bile into the stomach is an element in the causation of peptic ulceration — a theory which I first published in 1931.[2] The regurgitation of bile into the stomach as an active rather

than a passive element was first observed by Beaumont[3] (1833) on Alexis St. Martin, and reflux of pancreatic juice as a cause of ulcer formation by Stuber[4] (1914).

In a short communication[5] to the Physiological Society in Cambridge in 1954 I explained the function of the alimentary sphincters as a corollary of the Law of Isoperistalsis (postulated in 1931), and stated that they are adaptations to function and act *passively* as anatomically undifferentiated structures in the transmission of the peristaltic wave, and *actively* as functionally specialized structures in the prevention of reflux of the intestinal contents at the critical junctions.

It is interesting to note that Cannon and Blake[6] (1905) observed that the gastric component of a gastro-intestinal stoma hypertrophies and acts as a valve, allowing exit from, but preventing regurgitation into, the stomach. Truesdale[7] (1915) showed that gastro-enterostomy produced atrophy of the pyloric ring. Horwitz, Alvarez and Ascanio[8] (1929) confirmed this observation.

The ulcer problem, however, is not quite as simple as it may appear to be. In order to understand the pathogenesis of ulcer one must first take into consideration the rôle of the acid factor as an element in its causation. It is not involved in the production of the acute lesion but intervenes in the chronic ulcer. It is of fundamental importance to recognize that the acute ulcer never becomes chronic and that the chronic ulcer has never passed through an 'acute' stage. The chronic ulcer represents the final breakdown of the inflammatory reaction set up by the persistence of the irritating agent. Indeed, the acute and the chronic ulcer are two entirely unrelated disease entities.

In respect of the pathogenesis of the chronic lesion two distinct categories must be differentiated: (*a*) the one which occurs in the alkaline medium, namely, the oesophageal, anastomotic and Meckel's diverticulum ulcers, and (*b*) the one which occurs in the strictly ulcer-bearing area. The former is produced by direct action of hydrochloric acid on the alkaline mucous membrane (Mann and Williamson[9] have shown that if the duodenal mucosa is exposed to gastric juice without the admixture of the alkaline duodenal juices, an ulcer will form which is invariably situated on the intestinal side of the stoma). The latter cannot be produced by acid alone since it is inconceivable that the acid would destroy the glands which secrete it. A second element must, therefore, be concerned in association with hydrochloric acid in the production of the lesion and its persistence. There is strong evidence which incriminates bile as the second element involved.

My monograph[10] examines in great detail the various facets of the peptic ulcer syndrome and integrates them to form a 'concept' which I believe solves the peptic ulcer problem.

J.-Jacques Spira

59 Chesterfield House
London, W.1
28 October 1960

1. du Plessis, D. J. (1960): S. Afr. Med. J., **34**, 101.
2. Spira, J.-J. (1931): *The Causation of Chronic Gastro-duodenal Ulcer. A New Theory*. London: Oxford University Press.
3. Beaumont, W. (1833): *The Physiology of Digestion*. Plattsburgh: F. P. Allen (2nd edition 1947).
4. Stuber, B. (1914): Exp. Path. Ther., **16**, 295.
5. Spira, J.-J. (1954): J. Physiol. (Lond.), **125**, 27.
6. Cannon, W. B. and Blake, J. B. (1905): Ann. Surg., **41**, 686.
7. Truesdale, P. E. (1915): Surg. Gynec. Obstet., **26**, 298.
8. Horwitz, A., Alvarez, W. C. and Ascanio, H. (1929): Ann. Surg., **89**, 521.
9. Mann, F. C. and Williamson, C. S. (1923): *Ibid.*, **77**, 404.
10. Spira, J.-J. (1956): *Gastro-duodenal Ulcer. Physio-pathology, Pathogenesis and Treatment*. London: Butterworth.

GUEST LECTURER IN DERMATOLOGY

To the Editor: The Dermatological Group of the Medical Association of South Africa last year started a new and rather unique venture. It invited Dr. Geoffrey B. Dowling to be its guest lecturer for a period of about 2 months, during which time he travelled to all the large South African cities, made contact with various dermatologists, and attended dermatological clinics and meetings. He spoke to general practitioners and addressed the Annual Meeting of the Dermatological Group. Due to the unqualified success of this experience it was decided to extend similar invitations to a different dermatologist every year or two. I felt it to be a great honour indeed when I was invited to follow in the footsteps of so illustrious a predecessor.

The South African group thoughtfully chose March and April as the period for my visit. These are the most pleasant months of the year in that country, and my hosts were certainly not to blame for the fact that the political barometer was set at 'stormy'.

My way went through Pretoria, Johannesburg, Bloemfontein, Durban, Port Elizabeth, and Cape Town. In Pretoria, Johannesburg, and Durban there were dermatological case presentations of very high standard. The trip culminated in the national meeting of the Dermatological Group in Cape Town, in April, which included a number of excellent papers and well-chosen case presentations by the Dermatological Department of Groote Schuur Hospital.

There were many surprises. The first was that this turned out not to be a vacation, but a really intensive study tour for both sides! In every town there were meetings with local doctors, and the wealth of interesting clinical material gave me many opportunities, much appreciated, of learning something new and of seeing dermatology at work in a different country.

Outstanding among my dermatological impressions were those of the various vitamin disorders seen in the Bantu. Not only pellagra, of which there is a great deal, but also various protein deficiencies, including kwashiorkor. A most interesting fact was the appearance of red hair during the course of vitamin deficiency in Bantu children. The red hair blackens again after the vitamin disorder is cured. The relatively high number of cases of lipoid proteinosis is believed to be genetically traceable to one patient, who came from Holland with the disease many years ago. Also striking were the many cases of porphyria. This seems to be much more prevalent in South Africa than here in the Middle East, where sunshine is also certainly not lacking. Plantar hyperkeratosis, in a very painful and ulcerated form, is often seen among Bantus and a number of cases of congenital ichthyosiform erythroderma was presented. This was interesting, for this condition is only rarely seen in routine dermatological practice. True tropical dermatoses, such as chromomycoses, madura mycosis, and sporotrichosis, were also seen.

There is some interesting dermatological research going on in South Africa, such as that on primary pigmentation and on light sensitivity problems from biochemical and other aspects. Anatomical studies have also yielded new findings concerning the sweat gland apparatus.

On the whole there are few dermatological in-patient departments, and those do not have many beds. Unfortunately too, most of them are administratively connected with the medical department, and this may sometimes hamper their own development, in spite of the advantage of having good contact with internal medicine. Because of the few dermatological wards, and the high standards required of specialists by the South African Medical and Dental Council, there are relatively few dermatologists, and the number under training is also relatively small for such a big area.

But the most pleasant personal surprise for every dermatologist coming to South Africa will be the hospitality of his colleagues, no matter where he finds himself. It was unsurpassable. I do not know whether the members of the Dermatological Group of South Africa enjoyed the visit of their guest lecturer, but I greatly enjoyed being with them. I can assure any future guest that he is in for a memorable experience, and that he will surely profit from the rich dermatological material so generously spread before him.

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